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THE BRITISH JOURNAL OF CHILDREN'S DISEASES.

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THE

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OF

CHILDREN'S DISEASES.

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Original Articles.

THE SEVERE BLOOD DISEASES OF CHILDHOOD: A SERIES OF OBSERVATIONS FROM THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

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PART I.

Introduction.—The diseases of the hæmopoietic system, in spite of the great amount of work which has been expended in their elucidation, are still obscure as regards both their origin and their classification. If this is true of these diseases as they occur in the adult it is even more true in regard to the anæmias of children below the age of puberty. It has appeared, therefore, to us to be worth while to review and endeavour to classify as carefully as we are able the anæmias which have been met with in the wards of Great Ormond Street in the last two and a half years.

Before entering on any description of the diseases with which we propose to deal, there are a certain number of points which appear to demand discussion and definition in order to make plain our meaning and the general trend of our ideas.

Definition of anæmia.—The physician uses the term "anæmia" chiefly

in a purely clinical sense, judging its existence usually by the pallor of the skin and mucous membranes. If we try to define the term more accurately, anæmia is the failure of the blood-forming tissues to maintain the normal supply of corpuscles and of hæmoglobin to the rest of the body, and the gauge of its severity is measured by the failure of the capillary blood to reach certain normal standards in respect of numbers of corpuscles and amount of hæmoglobin. For the purpose of this clinical study we assume without discussion that after birth the hæmopoietic tissue of the body is the marrow contained in the bones of the skeleton, and that this is the source not only of the red corpuscles and the hæmoglobin, but of the white corpuscles as well. The exact site of formation of hæmoglobin is, we understand, unknown, but there can be no question that it enters into the red corpuscle in the bone marrow. Anæmia is therefore caused by (1) the destruction of the hæmoglobin and the erythrocytes more rapidly than they can be replaced by the normal or enhanced activity of the bone-marrow ; or (2) by the failure of an abnormal bone-marrow to produce sufficiently to maintain the normal quantities. In other words anæmia may be due to exaggerated extra-bone-marrow destruction, with normal or even increased bone-marrow production, or to failing or diseased bone-marrow production. The anæmia caused by severe hæmorrhage is a simple instance of the first type ; that of anæmia of the so-called " aplastic " form, or of leukæmia, belongs to the second division.

Methods of investigation.—The methods of investigation employed in the elucidation of the problems of anæmia are all well known ; there has been no considerable addition to them for many years, nor will there be in our opinion real advance towards the solution of the problems until we learn to use new methods of approach ; and it is one of the aims of this review to reconnoitre the ground anew and to survey the lines of attack.

Hitherto most study has been devoted to an attempt to understand the nature of the abnormal reactions of the bone-marrow by minute and painstaking examination of the nature and character of the cells produced in disease conditions. The first success was achieved more than eighty years ago by Bennett and Virchow, when by such study of cells they were able, independently and almost simultaneously, to separate the group of leukæmias from the other anæmic diseases. Elaboration of the method followed, and the work of the next half century has made us acquainted with the characters of the bone-marrow reaction in a number of conditions which have as one of their chief features the appearance of anæmia. But we venture to think that the profitable limits of this mode of investigation have been reached, and that the examination of the capillary blood by means of numerical counts, estimations of the proportionate numbers of white cells, and their examination by elaborate and careful

staining, though still indispensable, is unlikely to open a new vista on the problems. In this particular field of research we have learnt much of morphology, but very little of function. We know, for instance, that to many of the pyogenic infective agents the bone-marrow reacts by an abundant production of polymorphonuclear leucocytes, but we also know that in some instances this reaction is replaced by an unexpected lymphocytic increase—a phenomenon at present quite without explanation. Similarly there is as yet but little knowledge of the function or the causes of increase of the eosinophil cells, and still less of those of the basophil or large mononuclear cell.

Morphology of the leucocytes.—The morphology of the various cellular elements of the blood has been more especially studied in the leukæmic diseases, and an immense amount of painstaking work has been devoted to the relationship between the different forms of marrow-cell which make their appearance in the capillaries in these diseases. The fully developed myelocyte, which in health rarely, if ever, makes its appearance in the capillary blood, may in the more chronic, and even in some of the acute, forms of leukæmia be present in such numbers that it predominates over all other forms. In the leukæmias of children the fully developed cell, though often present, more usually gives place to a cell which is often termed a "lymphocyte." But it is not the normal lymphocyte of the capillary blood; it is larger, with more protoplasm which is often vacuolated, and a large, sometimes slightly notched nucleus which often takes the stain with a lighter tint of blue than the normal lymphocyte. This cell has received a great variety of names: premyelocyte, promyelocyte, myeloblast, and primitive cell, but it is generally admitted that it has its origin in the bone-marrow. The precise relationship of this cell with the myelocyte appears to us to be of little clinical importance, since it is admitted that its appearance in any number in the capillary blood is good evidence of bone-marrow leukæmia. Clinically, its only significance otherwise is that in those cases in which it predominates the course of the disease tends to be shorter and more acute than in those in which the fully developed myelocyte preponderates. The presence of either cell in large numbers in the capillary blood suffices to establish the clinical diagnosis of leukæmia. To this rule there is, however, one possible exception. We have said above that the myelocyte rarely, if ever, appears in the normal capillary blood. We may add that its appearance, save in the leukæmias, in disease is most exceptional. It is occasionally seen in the acuter stages of diphtheria, and occasionally a single cell is observed in other diseased conditions. But in the disease originally described by von Jaksch—anæmia pseudo-leukæmica infantum—it is always present; and it is occasionally difficult, because of this combined with some other

clinical features, to separate at once this disease from leukæmia. The distinction is clinically of importance from the point of view of prognosis, since more than half of the patients affected with von Jaksch's disease recover perfectly, whereas the leukæmic patients without exception die. The relationship of the two diseases will be further discussed later.

Oxydase reaction.—Closely connected with the question of the morphology of the blood-cells is the question of their chemical reactions. Some years ago, when the controversy about the origin of the "lymphocyte" was at its height, Schultze suggested that an examination of the chemical reaction of their contents might be useful to determine their origin. It was held that the presence of oxydase was sufficient to prove the bone-marrow origin of the containing cell, and various stains have been devised to demonstrate its presence. So far as these methods have tended to strengthen the contention of the supporters of the bone-marrow origin of nearly all forms of leukæmia they have been useful, but now that this view has won wide acceptance on this and other grounds the demonstration of the oxydase reaction in the blood-cells is no longer of great value. Further it must be recorded that there occur cases in which, although there was every reason to believe that the bone-marrow was the source of the abnormal cells, these failed to show the presence of oxydase. More recently Marfan, Minard, Saint Girons and Brousolle in France, and C. S. Graham in America have studied the peroxydase reaction, but of this we have no personal experience.

Bacteriology.—Both during life and after death bacteriological examination of the blood, pathological fluids and tissues has given us little of positive value. Organisms have been isolated, occasionally during life, and more frequently after death in several types of the severe anæmias; especially in the more acute leukæmias streptococci and diphtheroids have been found, usually towards the end of life or in post-mortem cultures. It is not, we believe, maintained by any pathologist that such organisms have any causal relationship to the diseases.

In the case of von Jaksch's disease protozoal bodies were described many years ago, but the discovery has never been confirmed. There is no other evidence to connect any of the anæmias of children with known protozoa.

Yet the nature, course and morbid anatomy of many of these forms of disease suggest strongly that their origin is to be found in some focus of infection. In the adult it is, we believe, accepted that an infective process may be the cause of an anæmia of the most severe type, indistinguishable as regards the reactions of the bone-marrow from the classical pernicious anæmia, and yet capable of recovery by removal of the infective focus. It is not, of course, essential that the infection should actually be localised

in the hæmopoietic system; it seems to be more likely that toxins, specific for the bone-marrow, are manufactured in foci remote from the site of their activity. We know already the rapidity with which an anæmia makes its appearance in, for example, the infection with *B. diphtheriæ*, and we have already referred to the fact that in this disease the bone-marrow occasionally reacts by sending a few myelocytes into the capillary blood, and the polymorphonuclear response to the pyogenic infections, wherever situated in the body, is an established fact. What we do not know is the mechanism by which the demand is conveyed to the marrow, though in the last instance the response is so rapid that it is difficult to escape from the conception of a substance manufactured by bacterial activity circulating directly to the bone-marrow and stimulating there a specific activity. Argument by analogy may, we know, be unsound, but analogy may well serve as the scaffolding for an hypothesis, and to us it would appear that the hypothesis of an infective origin for all the severe anæmias, including the leukæmias, is that which is most likely to prove fruitful. But the attempt to substantiate this, or indeed any other hypothesis, must be made with new weapons; it is, we think, clear that neither in the direction of research into the morphology of the blood-cells, nor by the current methods of the bacteriologist are we likely to come into possession of new facts.

Histology.—Nor is it probable that we shall get new light from histology. It is true that within the last twenty years the histology of lymphadenoma has been so accurately defined that we have in this method a means by which we can with certainty recognise, at any rate after death, the nature of the disease, even though the clinical signs have misled us. Yet as regards the leukæmias, and some of the other grave anæmias, the examination of sections of the organs and even of films of the bone-marrow may still leave us doubtful. It has been stated, for instance, that the kidney tissues in leukæmia invariably show a marked infiltration with the abnormal cell. In our experience, though it is often true that the kidneys show profound alteration by reason of the presence of the "leukæmic" cell, yet we have in our record several cases in which, from a consideration of the changes in the kidney, or even in the liver, it would be impossible to recognise with any degree of certainty the existence of leukæmia, and there are even uncertainties with regard to the changes in the bone-marrow. Films of the bone-marrow in cases of "aleukæmic leukæmia" may bear a striking resemblance to those obtained in cases in which there was grave anæmia, but no indication of leukæmia. Histological examination is, therefore, a valuable means of confirmation, or even of help in classification, but is not likely to give us new light on the problem of causation.

Hæmolysins, etc.—In the last few years a great amount of work has been done in respect of the properties of the serum of the blood. Yet we believe that in the diseases of the hæmopoietic system there has been little or no research in this direction. Yet we are in possession of certain facts which appear to point to the probability that blood serum contains substances which may be of importance both as regards the circulating blood and the hæmopoietic tissues. It is known that the injection beneath the skin of normal serum, especially of human serum, has a remarkably curative effect in the hæmorrhagic disease, melæna neonatorum, and the same power of controlling hæmorrhage from the mucous surfaces is sometimes observed in cases of purpura hæmorrhagica. The writers have further from time to time treated other forms of severe anæmia with subcutaneous injections of human serum, and in one instance, a case of von Jaksch's anæmia of great severity, with remarkably favourable effects. In two cases of leukæmia the injections appeared to have a beneficial effect on the course of the illness, though there was certainly no curative effect.

On the other hand, transfusion of the whole blood has not in our hands appeared to be of any benefit to the patients, even temporarily, and may even be harmful. We have examined the sera of several patients for the existence of hæmolysins, mostly with negative results. However, in two severe cases of purpura the patient's serum was found to hæmolyse an observer's washed erythrocytes, though it had no such effect on the patient's own corpuscles.

Our observations in this field are at present very few, and it is a method of research which demands more training and probably more knowledge of bio-chemical problems than we possess, but we do not doubt that in this direction light upon some of the problems of anæmia will come.

Coagulation time : fragility of the red corpuscles.—Two other methods must be mentioned which we have employed in some of our cases. The estimation of the coagulability of the blood is an extremely difficult matter, chiefly because the methods hitherto devised are insufficiently accurate. It is fairly easy to detect wide variations from a normal period, but it is, we believe, impossible with our present methods to obtain accuracy in the measurement of the smaller degrees.

The fragility of the red corpuscles to normal and subnormal saline has been extensively studied, and it can be stated with certainty that except in acholuric jaundice any marked increase in fragility is exceptional. In the severer grades of anæmia, and notably in those of the pernicious type, the red corpuscles, so far from being unduly fragile, are usually unduly resistant. The cause of these variations in either direction from a standard which is so extraordinarily constant for all ages is unknown,

but it is to be sought in the corpuscle and not in the serum in which they are bathed ; on the contrary experiments seem to point to the serum having a protective influence against the hæmolysis of the corpuscle.

The method we have used compares the hæmolysis of the red corpuscles of the patient with that of a normal individual in a series of dilutions of normal saline, beginning at ·8 per cent. down to ·3 per cent. with intervals of ·05 per cent. In the normal individual hæmolysis first occurs in the dilution of ·47 per cent.

Summary.—From this brief review it will be evident that for the investigation of the anæmias we are sadly in want of new methods. The examination of the morphology of the blood-cells has contributed most largely to our knowledge in the past, but has, it appears to us, reached a dead end—that is in respect of the advance of knowledge, for its application is still indispensable for the proper classification of the anæmic diseases. The examination of the fragility of the red corpuscles enabled Gilbert and Chauffard to define and recognise one peculiar form of anæmia, for which we appear to have a cure in splenectomy, although we do not yet understand the nature of the disease. With these exceptions no other method employed has added materially to our knowledge, nor appears likely to do so. The study of the nature and properties of the blood serum, including the mineral contents, seems to be most likely to be profitable.

Banti's disease.—Whether Banti's type of splenomegaly occurs at all in children under puberty is a matter of dispute, but that it is excessively rare is agreed, and the majority of the cases reported in the past under this title would probably not be admitted as such. Even in Italy a recent writer (Canelli, ' La Pediatria,' September the 15th, 1921) on this topic knows only of thirty cases, and points out how often in the pre-Wassermann days the factor of syphilis in the production of a splenomegalic anæmia was unrecognised. In our own experience we have not met with any case in which the history, course and symptoms were unmistakably those which are familiar in adults, those instances in which the diagnosis has been suggested almost invariably in the subsequent course of the disorder being classified under some other designation. At the same time it is fairly certain that the disease does sometimes begin before puberty, for the histories obtained from adults or adolescents point unmistakably in that direction. It remains, however, certain that many of the cases formerly reported under this heading would now be recognised as syphilitic, tuberculous, or belonging to the acholuric jaundice group.

Blood-platelets.—The *rôle* played by these structures in the normal individual is still imperfectly understood. The prevailing opinion is that they are connected intimately with the processes of coagulation. The following facts are known : Their numbers average about 200,000

to 300,000 per c.mm. and vary in health with great rapidity. In disease they may be increased or diminished to an extreme degree in either case, rising up to 2,000,000 or sinking to as little as 2000. Experimental work has shown that their place of origin is the bone-marrow, and hence it would be natural to expect that in the hæmopoietic disorders great variations would be met with. And, in the broad sense, this is the case; an increase is observed in those diseases in which there is a great activity of the bone-marrow, as for example the myelocytic form of leukæmia; and a decrease in those diseases in which there is reason to think that the hæmopoietic function of the bone-marrow is inactive or absent. The most marked decreases recorded have been observed in purpura hæmorrhagica, and the so-called "aplastic" anæmia. Experimentally, poisons such as benzol and bacterial toxins are found to diminish the numbers, but these same poisons when given in minimal doses appear to stimulate their production in a high degree.

The effects of these variations in number are uncertain. The researches of Ledingham, Bedson and others tend to show that a marked diminution is often associated with a tendency to capillary hæmorrhages, but that the absence or diminution of the platelets is not in itself the efficient cause of such hæmorrhages; there must be in addition damage to the endothelial lining of the capillaries.

To sum up: The known association of the platelets with the processes of coagulation, the marked diminution of their numbers in such diseases as purpura hæmorrhagica, and the experimental work all seem to point to a strictly limited function for the platelets in connection with the escape of the formed elements of the blood from their containing vessels. There does not appear to be anything to suggest that, except in this respect, the platelets are concerned either in the production or the cure of anæmia.

We have considered the cases which have occurred in this two and a half years under the following headings: (A) Congenital anæmias—acholuric jaundice, polycythæmia; (B) Severe anæmias—anæmia gravis, von Jaksch's anæmia (pseudo-leukæmica infantum); (C) leukæmia; (D) simple anæmia—chlorotic type; (E) lymphadenoma; (F) purpura.

Congenital anæmia.—It would seem probable that just as other tissues of the body are subject to congenital malformation, so the tissues of the hæmopoietic system should occasionally suffer in this fashion. But, with one exception, presently to be discussed, there is no evidence available in support of this hypothesis. Even in the rare cases where anæmia exists at the time of birth, or shortly afterwards, the causes which produce the anæmia appear to be those which are familiar in later life, especially the septic processes arising from infection of the umbilical

cord. Nor in those children who are otherwise congenitally malformed does the blood appear to be, with our present knowledge, in any way abnormal in its constitution; the Mongols, for example, have the usual amount of hæmoglobin, and the usual type of red and white corpuscles. Only in the case of congenital pulmonary stenosis is there a marked abnormality, and here the abnormality is rather quantitative than qualitative. Melæna neonatorum, again, produces in the blood the expected changes—that is there is a marked reduction in the numbers of the red corpuscles, and in the amount of the hæmoglobin; and the bone-marrow reacts to the loss of blood by the production of erythrocytes in a rapid fashion, often with an excess of nucleated erythrocytes in the peripheral circulation.

Congenital acholuric jaundice.—The only type of anæmia of which we have any present knowledge, which can be fairly called congenital, is that associated with acholuric jaundice, an affection which is certainly in many cases an inherited disorder. Whatever is the exact pathology of the condition, its most prominent characteristic is the congenital fragility of the red corpuscles. The corpuscles are themselves well formed and do not in their morphology show any abnormality, but are more easily dissolved than the normal corpuscle. The fact is easily ascertained by comparing the resistance of the red corpuscles of such a case with those of a normal individual to the action of solutions of sodium chloride in a graduated series of dilutions. In the normal individual the corpuscles are not destroyed by immersion in a salt solution of a greater percentage strength than 0·47 per cent. This figure has been ascertained with precision by numerous workers, including Widal and Abrami. At this point hæmolysis of the corpuscles is slight; with a weaker solution, 0·4 per cent., it is already considerable, and with solutions weaker than this it is complete, no visible corpuscle remaining intact after a very short exposure to the solution. In strengths above 0·47 per cent. there is not the least trace of hæmolysis, the corpuscles retaining their shape and their hæmoglobin. In cases of congenital acholuric jaundice, on the contrary, the corpuscles break down and give up their hæmoglobin in solutions of sodium chloride as strong as 0·6 per cent., 0·7 per cent., or even 0·75 per cent. It is certain that the cause of this behaviour resides in the corpuscle itself, for if the serum and corpuscles be separated and the serum of the patient be added to normal corpuscles, properly washed, and these then examined as to their fragility, no abnormal hæmolysis occurs; whereas the serum of a normal individual similarly added to the washed corpuscles of a patient suffering from this affection does not reduce the abnormal fragility of his corpuscles. It is, therefore, obvious that the cause of the abnormal fragility must lie in the corpuscular structure.

The abnormal fragility is an inherited quality. It occurs in families and is transmitted from one generation to another. Two families are known in which it has appeared in four generations. Unlike hæmophilia it is not apparently either confined to one sex, nor transmitted by one sex only, but attacks males and females alike and is transmitted either by the father or the mother. Like hæmophilia, however, it is apparently partial in its distribution; not all the members of a family are affected, and even those who are affected suffer in different degrees. In some instances, although the abnormal fragility is present, the individual has never suffered any disability from its presence; in others, and these are the patients who attract our attention, there are constant attacks of severe anæmia and jaundice. The anæmia is sometimes severe enough to endanger life, and frequently to disable the patient from earning a livelihood. The attacks are definitely paroxysmal; at times the patient is apparently normal in colour, without jaundice, and his blood-picture almost normal; at other times, for no apparent reason, he is anæmic to a severe degree, and jaundiced. The urine rarely, if ever, in this disease contains bile-pigments but is usually deep-tinted with urobilin.

As in hæmophilia, apart from the cases occurring in known families there are seen cases of "bleeding" indistinguishable from family haemophilics, so in this disease there are frequent examples of the affection in individuals none of whose relatives are affected.

Associated with the abnormal fragility of the corpuscles is a remarkable clinical phenomenon—enlargement of the spleen. In all the cases which we have seen of this disease the spleen has invariably been large, and in some instances enormous, reaching to the mid-line and the iliac crest; in others it is, though enlarged, comparatively a small organ. Its enlargement varies with the degree of the severity of the affection; when the anæmia is at its height, the spleen is at its largest; when with rest and other treatment the anæmia improves, the spleen tends to become smaller. Yet even in cases where the disability is trivial, the tendency appears to be for the spleen to increase in size as the patient grows older. There are not a few cases in which, apart from the recurring attacks of jaundice, there is no disability at all, the patient being able to get about and work hard, even at manual labour, with no further symptoms and with a normal cell count and normal hæmoglobin. During the attacks of jaundice the liver may be found to enlarge, and later, after the attack, to recede.

The case recorded in our list corresponded in most essentials with the foregoing description, except that he was the only member of his family affected. The attacks of anæmia began to affect him severely at the age of $2\frac{1}{2}$ years, and on one occasion at the hospital his hæmoglobin had sunk to less than 20 per cent. and his red blood-corpuscles to less than $2\frac{1}{2}$

millions. His fragility was constantly about 0·55–0·6 per cent. of salt solution. His spleen was considerably enlarged, though not enormous, and varied in size with the severity of the anæmia and the jaundice.

It is an ascertained fact that if for any reason an individual loses his spleen—for example, if it be removed after rupture—the resistance of the erythrocytes to dissolution in salt solution is definitely increased. Whereas in a normal individual hæmolysis takes place in solutions weaker than 0·47 per cent., in such a man hæmolysis may begin only in a solution of 0·3 per cent. The relationship of these phenomena is unknown. Whatever it may be the removal of the spleen in cases of acholuric jaundice is an obvious suggestion, and the results of such removal are in our experience most excellent. After the removal of this patient's spleen his condition at once materially improved, and though he had a recurrence of anæmia, this time without any jaundice, about two months after the operation, four months later he had a normal fragility of the red corpuscles and over five million red corpuscles with 0·65 per cent. of hæmoglobin. Other cases of the same kind similarly treated have responded in the same fashion, and though the explanation of the mechanism is not obvious it is clear that in splenectomy we have a means of removing these individuals from the category of chronic invalids. Not all the sufferers from acholuric jaundice will require such an operation, for many of them are, apart from their occasional yellowish colour, in excellent general health and experience no material drawback from their inherited abnormality.

It is, we think, necessary to point out that in cases of jaundice with splenomegaly and recurrent anæmia the simple examination of the fragility of the red corpuscles will sometimes enable the physician to offer the patient a hope of permanent relief, and that the omission of this examination has in the past led to not a few errors of diagnosis.

An unusual case of polycythæmia.—The remaining case, which we have provisionally placed in the category of congenital anæmia, is, so far as our knowledge goes, unique.

The child was admitted for anæmia, diarrhœa and wasting, and on examination proved to have a consistently high erythrocyte count. In many examinations extending over more than a year she has never been found with less than 6,000,000 erythrocytes per c.mm., and often with 8,000,000 or 9,000,000. Her hæmoglobin, on the other hand, has never been above 90 per cent. of the standard and has been as low as 65 per cent. She has a large soft spleen, and marked ricketty changes in the bones. We have never been able to satisfy ourselves that she has a congenital cardiac lesion, and the cause of her erythræmia remains obscure. Meanwhile her health is on the whole good and her ricketty lesions are disappearing. She is seldom cyanosed, and at her best looks a normal healthy child.

Examples of Acholuric Jaundice.

Case 1.—A girl, aged 6 years, was admitted under Dr. Hutchison on October the 11th, 1921, with a history of being yellow from birth. An eight months' child, she was noticed

to have jaundice on the tenth day after birth, and this has persisted ever since. Her only complaint was pain in the groins on walking far. She was breast-fed for two months and then had Nestlé's milk. The mother was twice married, her eldest daughter by the first husband was normal and the fragility of the red blood cells was normal. The first child of the second marriage died at thirteen months of pneumonia and was always pale and had a large abdomen. The second child was the patient.

The father was pale and the red blood cells showed a slight increase of fragility.

A cousin of the father's had her spleen removed twenty years ago for anæmia, and the fragility of her red cells is definitely increased at present.

The patient was a well-nourished child, of a pale yellow colour. There was slight clubbing of the fingers, the spleen reached half an inch below the level of the umbilicus and was very hard; the liver reached three fingers' breadths below. The heart was normal except for a hæmic systolic murmur at the base. The urine was brown and contained no bile. The blood count was as follows: Red cells, 2,300,000; hæmoglobin, 30 per cent.; colour index, ·6; white cells, 10,800; polymorphs, 66 per cent.; small lymphocytes, 31 per cent.; large lymphocytes, 3 per cent. The fragility of the red cells was extreme.

The spleen was removed on November the 10th, 1921, and four days later there was no change in the fragility of the red corpuscles.

A section of the spleen showed diffuse fibrosis with giant-cell formation, and these cells contained red blood-corpuscles.

Case 2.—A boy, aged 6 years, was admitted under Dr. Poynton on January the 1st, 1922. At the age of eight and a half months he was noticed first to have a strange lemon colour. For the last four months he has been having attacks of sickness followed by diarrhœa. During these attacks he is said to be delirious. When an infant he was diagnosed as splenic anæmia. The family history throws no light on the condition. He is a pale, waxy, well-nourished boy. There is no clubbing of the fingers, but there is bossing of the skull and beading of the ribs. The spleen reaches to within two fingers' breadths of the umbilicus and the liver is four fingers' breadths below the costal arch. The heart is normal except for a hæmic systolic murmur. The urine was brown, but did not contain bile. Wassermann negative and Pirquet reaction negative. Blood examination, January the 7th, 1922: Red cells, 1,506,000; hæmoglobin, 20 per cent.; colour index, ·6; normoblasts, 4 per 100 white cells; white cells, 15,000; polymorphs, 71 per cent.; small lymphocytes, 21 per cent.; large lymphocytes, 6 per cent.; large mononuclears, 2 per cent.; fragility of the blood was moderately increased up to and including ·6 per cent. saline. A month's stay in hospital, in spite of treatment, has produced no improvement in the blood picture and the fragility of the red blood cells. The boy seems slightly improved and the question of splenectomy is still under consideration.

Case 3.—Boy, aged $2\frac{3}{4}$ years, admitted under Dr. Hutchison, September the 28th, 1921. The child had suffered from bilious attacks and jaundice from eight months of age. He was breast-fed for eleven months. There was nothing in the family history to throw any light on the case. These bilious attacks consist of bouts of vomiting preceded by jaundice which increases in depth and becoming green at the height of the attack and gradually fading. For about a week after this the child is pale. The vomit is clear green with some slime. Preceding the attack there is langour, feverishness and anorexia, together with enlargement of the submaxillary glands. During the attacks the urine becomes a deep yellow colour. Between the attacks the child seems well.

A fat, flabby child, submaxillary and cervical glands palpable. Spleen enlarged and firm, extending down to the level of the umbilical plane. Liver enlarged, one and a half fingers' breadths below costal margin. The urine is a deep yellow colour, acid, urates, acetone and bile present. This observation on the presence of bile was not confirmed at subsequent examinations. Wassermann test negative.

Blood examination: Red cells, 3,870,000; hæmoglobin, 50 per cent.; colour index, ·6; white cells, 17,600; polymorphs, 70 per cent.; small lymphocytes 26 per cent.;

large lymphocytes, 2 per cent. ; large mononuclears, 2 per cent. ; normos, 3 per 100 white cells. The fragility of the blood was slightly but definitely increased.

First blood count, September the 29th, 1921 : Red cells, 4,127,000 ; hæmoglobin, 35 per cent. ; colour index, ·4 ; normos, 4 per 100 white cells ; white cells, 18,6000 ; polymorphs, 70 per cent. ; small lymphocytes, 19 per cent. ; large lymphocytes, 7 per cent. Treated with iron and left hospital in a slightly improved condition.

Case of Polycythæmia.

Girl, aged 3 years and 4 months. First came under observation when two months old, when she was brought to St. Bartholomew's Hospital, under Dr. Thursfield, for swollen feet. Her colour was not at that time remarked upon.

When a year and eight months old, she was admitted to Great Ormond Street with wasting and diarrhœa, which had been more marked for the past four months.

On admission she was seen to be markedly rachitic and her breathing rapid and grunting. It had always been noisy and remained so. The child had well-marked diarrhœa, and was a slightly cyanosed colour. Her spleen was well below the costal margin.

During the next ten weeks several blood counts were done and they seemed to vary with the degree of diarrhœa, but were always above 6½ million red cells, and rose above 9 million on one occasion. The white count fell from 38,000 on admission to 12,750 on discharge, and the proportions were normal. The hæmoglobin remained just below 90 per cent.

She has been seen at intervals since and her blood count has always shown more than 6½ million red cells. In April, 1922, the spleen was still three fingers' breadths below the costal margin ; she was slightly cyanotic in colour, especially so on exertion. Her rickets had almost entirely disappeared, and her blood count showed 6½ million red cells with a hæmoglobin count of 80 per cent. Her white cells were 10,750, and the proportion of the white cells was normal.

Her Wassermann reaction has been negative on more than one occasion, and there is no clinical evidence that she has congenital heart disease. The family history does not help in this case in the least.

Anæmia gravis.—The most puzzling type of anæmia is that which has received a number of different names, but is perhaps most widely recognised under the title of aplastic anæmia. For many reasons this is an undesirable name, but especially because there is in many severe anæmias an aplastic stage, and because the disease which we are now considering includes some instances which could not be reckoned as aplastic in type. It is probable that in this category there is more than one type, but the signs which may serve to distinguish them have not yet been clearly discerned. In some cases the symptoms and the course of the disease and the pathological appearances remind us of leukæmia, although the blood, when studied microscopically, has not the characters of the fully-developed leukæmia : in others the clinical phenomena appear to bring the disease within the conception of the purpuras ; in others again the resemblance to the classic type of idiopathic pernicious anæmia makes us forget for the moment the striking differences ; and yet in others we seem to be dealing merely with severe types of secondary anæmia.

We have preferred to use the name of anæmia gravis for this group, and have recorded three cases under this heading with the full conscious-

ness that in each of them there is something to be said for their inclusion in some other group; for instance, Case 1 might be considered as a purpura, and Case 3 as an example of an atypical leukæmia; but after considering these possibilities these three cases seem to us to stand by themselves as examples of a form of anæmia not very uncommon in children, which holds the place in the diseases of children which is occupied by the pernicious anæmia of adults.

When we examine these cases in detail we find that all three were children of eleven years of age, and this is the first point on which we lay stress. The type of anæmia which we are now considering does not occur in infants and is seldom seen before seven or eight years of age, and more usually a year or two later. Among eleven such cases seen by one of us the youngest was aged seven and all the others were over ten years of age.

Secondly the history of the patient, unlike that of most other types of anæmia, usually dates back many months or even years. In these three cases the patients had been ailing, or there had been hæmorrhage or pallor for six months, a year, and seven years respectively, before their condition became really serious. The same feature has struck us in other cases of a similar type, whereas in leukæmia, purpura, lymphadenoma, and "school-age" anæmia it is unusual, we think, to obtain a history antedating the serious condition by more than a few weeks. These diseases when present generally give rise to some impressive phenomenon entailing medical attention early in their course. In this group, on the contrary, the failure of health is so gradual that although it is noted it is assigned to some passing indisposition and for many months it is not taken seriously.

When we pass to the consideration of the clinical symptoms, the most striking feature is the extreme degree of the anæmia. The hæmoglobin is always less than 30 per cent., sometimes less than 20 per cent., and the red blood-corpuscles reduced to half their normal number or less. The result is a transparent pallor of the skin and mucous membranes which is most striking. With this diminution in the erythrocytes and the hæmoglobin is usually associated a marked diminution in the numbers of the white corpuscles, a diminution which seems to take place chiefly, though not exclusively, at the expense of the polymorphonuclear leucocytes. Thus in the three cases in our list, Case 1 had 3,400 leucocytes, of which 2,000 were polymorphonuclear; Case 2 had 4,400, of which again a little more than 2,000 were polymorphonuclear; and Case 3 had 5,200, of which 1,800 were polymorphonuclear. The normal figure of the polymorphonuclear leucocytes at this age lies between 5,500 and 7,000.

Although this feature is usually present, there are occasional exceptions; in one case seen recently there was a well-marked polymorphonuclear

leucocytosis. It is of interest to note that in this instance recovery from a desperate condition was unusually rapid.

There is, of course, as in all severe anæmias, much poikilocytosis, and in some cases there appear nucleated red corpuscles in some numbers, and though in these three cases no myelocytes were observed, we have seen them occasionally in similar patients, though never reaching 1 per cent. of the total leucocytes. Other abnormal forms are not observed.

The next point of interest is the occurrence of hæmorrhage. Extensive or extreme hæmorrhage is very unusual; nothing like the bleeding seen, for instance, in purpura hæmorrhagica; yet in every case there is a marked tendency to bleed; epistaxis in the first case; intestinal bleeding in the second; and subcutaneous and submucous hæmorrhages in the third. Retinal hæmorrhages also are common, as indeed in most severe anæmias. The point on which we desire to lay stress is that hæmorrhage of any degree is rather an accident and does not take a prominent place in the symptomatology of this disease, as it does, for instance, in purpura hæmorrhagica and in some cases of acute leukæmia.

The liver and the spleen are always enlarged, but the latter is often of quite moderate size. The superficial lymphatic glands are not enlarged, or if occasionally the cervical glands are palpable the degree of enlargement is no more than is usually met with in children of the type of hospital patient. The tonsils also may be increased in size, but again no more than is common. The large septic bleeding tonsils of some cases of purpura and leukæmia are not seen.

The course of the disease is variable. The larger number of the patients drift steadily downwards and die of exhaustion; in some cases the red cell count sinks below half a million per c.mm.; in others it remains somewhere about two million till death. In the patients who recover, convalescence is slow, and the restoration of the blood to normal may take many weeks or months; the most favourable sign in this repect is, we believe, a steady increase in the polymorphonuclear leucocytes.

Post mortem there are the usual appearances connected with severe degrees of anæmia, but not as a rule anything distinctive. The condition of the bone-marrow is of importance, but unfortunately our observations on this point are too few in number to allow of any generalisation. In all the cases which we have seen the bone-marrow has been white, not in the least resembling that of the pernicious anæmia of adults. The liver in one case was of a chocolate tint and gave a strong Prussian blue reaction, but in the other case in our list it was merely anæmic and gave no blue reaction.

In Case 3 one kidney was the seat of numerous small abscesses, chiefly distributed in the cortex; during life a small quantity of pus had been

observed on one occasion in the urine about ten days before death, but on all other occasions none had been found. It does not seem probable that it was more than an accidental infection.

We conclude, therefore, that the disease from which these children suffer is a grave anæmia, which is not a leukæmia nor a purpura, which is not related to lymphadenoma, and has many points of difference from the pernicious anæmia of adults. It is not necessarily fatal, though the majority die. It is not really aplastic, since there is often evidence at some time or other during the course of the illness of bone-marrow activity. As regards its pathology we have sought in vain both during life and after death for any direct evidence of bacteria infection, and in this respect our experience resembles similar attempts in the pernicious anæmia of adults. Yet just as in that disease there is a conviction in the minds of many physicians that ultimately the cause will be traced to the presence of some focus of infection, so in these children we think that it is probable that there is some infective process at work producing the poisons which act so disastrously on the hæmopoietic system.

Diagnosis is in our view most difficult. Perhaps the greatest difficulty is to decide whether a given case is or is not an example of atypical leukæmia. We believe that there are cases of so-called "aleukæmic" leukæmia which present the same anæmia, the same leucopenia, and the same atypical course. But in such cases the differential count of the leucocytes is strikingly dissimilar. In such leukæmias the principal leucocyte present is the "lymphocyte," and as the disease progresses this cell tends more and more to predominate. In the disease which we are considering, on the other hand, the lymphocytes present are the normal lymphocytes of the blood and they are present in diminished numbers, though as we have pointed out, often less diminished than the polymorphonuclear cells. Moreover, as the disease progresses they do not tend to become more numerous. Even after death with an opportunity of examining the tissues, it is sometimes hard to determine whether there has been a leukæmia present; in some of the cases which clinically were typical acute leukæmia, there has been little infiltration of the tissues. But in all cases of clinical leukæmia the bone-marrow has been conspicuously red, quite unlike the white anæmic marrow of this group. Further, though in some cases of leukæmia the superficial glands are not enlarged, we have not seen any case in which the glands were not found enlarged or hæmorrhagic after death. It would appear that these two signs, the redness of the bone-marrow and the condition of the lymphatic glands, especially of the mesenteric group, afford the most certain post-mortem indications of the nature of the disease.

Supposing we have dismissed the diagnosis of leukæmia, we have still

to consider other possibilities. There are instances of purpura hæmorrhagica in which the anæmia and the leucopenia are as striking as in this group. Apart from the marked hæmorrhages which are so characteristic of this disease, and which form no part of the symptomatology of the disease under consideration, in the few cases which we have seen the bone-marrow has been red rather than white. It is, we believe, more difficult to separate the cases of acute purpura from those of acute leukæmia than to distinguish anæmia gravis from purpura.

Lymphadenoma is another disease which at times presents the same leucopenia and severe anæmia; we have twice known lymphadenoma proved post mortem to be that disease diagnosed clinically as "aplastic anæmia." The presence of enlarged glands in both instances, not only in the neck but in the axillæ, should have led to a more correct diagnosis.

Lastly there is the question of pernicious anæmia such as is seen in adults. That this disease does occur in children is possible, though it is certain that most of the cases reported under this heading are examples of other forms of severe anæmia. We have never seen a case in a child which conformed to the type of adult pernicious anæmia, though we have met with several in which that diagnosis has been discussed, only to be discarded either by means of accurate blood counts or when the post-mortem examination has revealed the true nature of the affection. This disease bears much resemblance to the pernicious anæmia of adults, but differs from it in two directions: first in the fact that while in pernicious anæmia there is usually abundant evidence of bone-marrow activity that is exceptional in anæmia gravis; secondly, in the true pernicious anæmia of adults there is, we believe, almost without exception a red marrow, whereas in this disease the marrow is white. From the purely clinical aspect there is nothing to separate the two conditions.

The prognosis of anæmia gravis is never good, for the majority go steadily downhill, and no treatment seems to be of much benefit to them. And yet now and then a patient who has nothing to distinguish his condition from that of the fatal cases proceeds to complete recovery. The patients whom we have seen recover have for the most part been treated with serum subcutaneously or intravenously. The serum may be either human or from the horse. Transfusion has in the few instances in which it has been employed appeared to be of little benefit.

Examples of Anæmia Gravis.

Case 1.—Boy, aged 11 years, admitted under Dr. Poynton, September the 21st, 1920. He had suffered from attacks of sickness and pain in the head for fourteen months. The family and previous histories threw no light on the case. In July and December, 1919, and April, 1920, he had attacks of vomiting and pains in the limbs, and in the December attack "yellow jaundice" for ten weeks.

On admission he had a lemon tint, and the heart was rapid with a basal systolic bruit. These were the only positive clinical signs. The blood count was as follows: Red cells, 3,260,000 per c.mm.; hæmoglobin, 25 per cent.; colour index, ·4; white cell count, 4400; polymorphonuclears, 58 per cent.; lymphocytes, 41 per cent.; eosinophils, 1 per cent.

He remained in the hospital with the same negative symptoms until Christmas. The outstanding feature was the failure of every remedy to produce a substantial improvement in the anæmia.

In October the red cell count was 2,810,000 per c.mm.; hæmoglobin, 25 per cent.; colour index, ·4. The white cell count, 6400 per c.mm.

In November there was no change.

In December there was some improvement: Red cell count, 3,172,000 per c.mm.; hæmoglobin, 30 per cent.; colour index, ·48.

The differential counts had shown a preponderance of polymorphonuclear cells.

In January he was out of hospital, and the improvement continued. Red cell count, 3,650,000 per c.mm.; hæmoglobin, 32 per cent.; colour index, ·4. The white cells had risen to 11,000 per c.mm.

In February the red cells fell to 2,670,000 and the white cells to 8600 per c.mm. He was readmitted on March the 9th, 1920, with dyspnœa, rheumatic pains and swelling of the feet. His face showed the same lemon colour. The red cell count was 1,580,000 per c.mm.; hæmoglobin, 20 per cent.; colour index, ·6; white cell count, 4200 per c.mm.; polymorphonuclears, 71 per cent.; lymphocytes, 27 per cent.; nucleated red cells were present, 2 per cent.

On April the 1st there was much improvement in the red cell count up to 4,106,000 per c.mm. About the 9th there was a sudden increase in the pallor, and the red cell count fell to 1,856,000 per c.mm.; hæmoglobin to 20 per cent.; colour index, ·4. At this time the spleen became palpable and the liver had increased in size, almost reaching to the level of the umbilicus.

In May there was increasing pallor and some vomiting. The blood count showed: Red blood cells, 2,750,000 per c.mm.; hæmoglobin, 20 per cent.; colour index, ·3; white blood cells, 7600.

In June all the symptoms of profound anæmia increased and the temperature rose and became irregular. At this time it was ascertained that the patient's serum did not agglutinate or hæmolyse his father's red corpuscles. The red cell count had fallen to 1,480,000 per c.mm., and nucleated red cells were present, 74 per c.mm. On June the 12th, 200 c.c. of the father's blood was transfused, but a large vomit followed, the temperature rose to 105·8° F., and the boy died next day.

The fragility of the red corpuscles was normal, the Wassermann reaction negative, and no ova were found in the fæces.

The necropsy was remarkably negative except for two features: (1) The bone-marrow was white. (2) In the lesser curvature of the stomach was a tumour projecting into the cavity about the size of a pigeon's egg, which proved on microscopy to be a fibromyoma. The mucous membrane over it was discoloured at two points, but there were no signs of bleeding from the mucous surfaces. There was no Prussian blue reaction in the liver or spleen, no cellular infiltrations in the viscera, and no valvular disease.

Case 2.—Girl, aged 12 years. Admitted under Dr. Thursfield, January the 22nd, 1921, for progressive weakness and anæmia since July, 1920. She was an only child and an orphan, losing her father in the war, and her mother from heart disease. She developed earache after swimming in the summer and never seemed to be well after this. Three times during December there had been bleeding from the gums. There was no history of sore throat.

The child was very pale, but there was no purpura or œdema. The heart was rapid with basal systolic murmurs. The cervical glands were easily felt and the liver and spleen were

both palpable. The gums were slightly œdematous, the tonsils healthy. The urine was normal, and there was no evidence of respiratory disease.

The blood count was as follows: Red blood cells, 1,190,000 per c.mm.; hæmoglobin, 30 per cent.; colour index, 1·3; megaloblasts, 2 per cent.; white blood-cell count, 5200 per c.mm.; polymorphonuclears, 35 per cent.; lymphocytes, 63 per cent.; eosinophils, 2 per cent.

The history in hospital was one of continual hæmorrhages; epistaxis, purpura, bleeding from the gums, and retinal hæmorrhages. On January the 27th a colony of streptococci was isolated from the blood. The temperature throughout was raised and irregular.

Blood 6 oz. was injected intravenously from the aunt, whose blood as donor was suitable. Antistreptococcic serum was also given. On March the 3rd albumin and pus were detected in the urine, and death occurred on the 11th.

The blood count on the 9th of March showed: Red blood cells, 625,000 per c.mm.; hæmoglobin, 10 per cent.; colour index, ·9; white blood cells, 2800 per c.mm.; polymorphonuclears, 66 per cent.; lymphocytes, 34 per cent.; the platelet count showed 34,000 per c.mm.

Autopsy.—The fat was golden yellow, muscles dark red, numerous subserous petechiæ were present. There was no organic heart disease. The liver was chocolate-brown and gave the Prussian blue reaction. There was some perisplenitis. The right kidney showed evidence of pyelonephritis. The bone-marrow was pure white and gave no Prussian blue reaction. There was no cellular infiltration of the viscera. The microscopy of the right kidney showed multiple abscesses throughout.

Case 3.—This patient, who has apparently recovered, was a boy, aged 11 years, admitted under Dr. Poynton, who gave a history of bleeding from the mouth and nose at intervals for the last four years. There was no hæmophilic pedigree.

When admitted to hospital he was very white, with pale lips, foul breath and carious teeth. His spleen was felt just below the costal arch. There were some petechiæ and functional cardiac murmurs. The temperature was irregular, between normal and 100° F.

The blood examination showed: Red cells per c.mm., 2,200,000; hæmoglobin, 20 per cent.; colour index, 0·48; white cells per c.mm., 3400; polymorphonuclears, 60 per cent.; lymphocytes, 40 per cent. Coagulation time normal.

The boy improved and a month later his red cell count had risen to 4,127,000 per c.mm. Carious teeth were extracted and the mouth was thoroughly cleansed. Improvement steadily continued until five weeks later. He was getting up when he commenced to relapse with increasing pallor and petechiæ on the arms and legs.

The blood count was now: Red cells, 2,980,000 per c.mm.; hæmoglobin, 28 per cent.; colour index, ·5; white cells, 5400 per c.mm.; polymorphonuclears and mononuclears, 50 per cent.

The spleen, which had diminished in size, now became again palpable. Enforced rest produced a gradual improvement which has been maintained.

Three months after leaving the hospital the blood count was as follows: Red blood cells, 4,678,000 per c.mm.; hæmoglobin, 75 per cent.; colour index, 0·8; white cell count, 7600 per c.mm.; polymorphonuclears, 74 per cent.; mononuclears, 26 per cent.

Later accounts from his parents show that he is keeping well.

Reviewing these three cases we first direct attention to the blood picture in Case 2, viz., red cells, 625,000 per c.mm.; hæmoglobin, 10 per cent.; colour index, ·9; white cell count, 2800 per c.mm.; differential count, polymorphonuclears, 66 per cent., lymphocytes, 34 per cent.

An analysis of this shows the extreme fall in the red count and hæmoglobin and leucopenia with a normal differential count. If a count such

as this—that is, one with all the evidence of a total failure in blood formation—were invariable, the widely-used name of "aplastic anæmia" would be a good one. If, however, we take Case 1 we find on the other hand very clear evidence of reaction and blood-formation during the illness. A low red count mounting rapidly to 4,000,000 per c.mm. cannot be claimed as an aplasia.

We realise that a disease with so many negative features as this is, however, likely to be defined in accordance with individual experience, and it is possible that the three examples given here may not be accepted as "aplastic anæmia," or, as we would term it, anæmia gravis, by other observers.

Case 1 may be claimed as a secondary anæmia due to overlooked bleedings from the tumour in the wall of the stomach. Case 2 as an anæmia resulting from a septicæmia the result of pyelonephritis, and associated also with a septic mouth. The third as anæmia resulting from a septic mouth.

We do not deny that an anæmia gravis may arise from all these causes, and others as yet unknown; indeed, it is our conception of this disease that it is secondary to some blood poison, infective or otherwise. If there is no demonstrable cause then perforce we are obliged to call it a primary anæmia gravis. And when we use this term we mean a condition different to leukæmia or chlorosis, or acholuric jaundice, etc. These cases show these differences, and in particular the white appearance of the bone-marrow is a most remarkable feature only met with in anæmia gravis. It will be noticed that the cases were children about 11 years, that the illnesses were slow and mysterious in origin. Also that the colour of the face might be icteric and the spleen slightly enlarged. Towards the end there was much dyspnœa, fever and general listlessness and weakness.

Case 1 showed striking remissions and his illness resembled closely an adult pernicious anæmia with its duration compressed from years into months.

Although writers have insisted upon the wide differences between anæmia gravis in childhood and pernicious anæmia in the adult, we are inclined to think that the essential resemblance is much more convincing, and to believe that the differences do not lie so much in the nature of the disease as in the fact that we are dealing in one case with an adult, in the other with a child.

There are rare examples of complete recovery in adult pernicious anæmia, and the third of our cases is interesting from this point of view.

The possibility of recovery makes the prognosis at the present time a very difficult one, and bearing upon this a remarkable case recorded by

Porter Parkinson in the British Journal of Children's Diseases, 1919, xvi, pp. 1–4, may be referred to.

A boy, aged 9 years, profoundly anæmic and puffy, with bleeding gums, sore mouth and petechiæ, but with no enlargement of the liver or spleen, was admitted to hospital with a blood count as follows: Red cells, 1,200,000 per c.mm.; hæmoglobin, 20 per cent.; colour index, 0.9; no nucleated cells; white cells, 800 per c.mm.; polymorphonuclears, 20 per cent.; lymphocytes, 72·5. There were numerous hæmorrhages. The boy went from bad to worse, and was transfused with citrated blood on two occasions, when alarming collapse developed, and he became comatose after the second transfusion and was taken home to die. There he remained incontinent and unconscious for a fortnight and then slowly improved. His red cell count is recorded as sinking to 580,000 per c.mm., and the leucopenia with great diminution in polymorphonuclear leucocytes had persisted. In January, 1919, eleven months later, he was in good health, with a blood count of 3,960,000 red cells per c.mm.; hæmoglobin, 65 per cent.; colour index, 0·8; white cell count, 5800 per c.mm.; polymorphonuclear leucocytes, 65 per cent.; mononuclears, 32 per cent.

From the blood counts it seems difficult to exclude a lymphocytic leukæmia, but the view held by Parkinson was that of an "aplastic anæmia" and the recovery would favour this. In any case this illustrates the remarkable recuperative powers that are possible in these grave anæmias, and which in this instance appeared to have developed under negative treatment at home, rather than active measures in hospital.

Such a case raises the question whether in aplastic anæmia there may develop a relative preponderance of lymphocytes in the differential count. This certainly occurred in our Case 2.

Our experience of leukæmia has been that the improvements have not been maintained, but an inquiry from Dr. Parkinson upon this point over a year later resulted in his expressing the belief that he would certainly have heard of any relapse in this case.

We have already commented upon the gastric tumour, pyelonephritic and septic mouths in our cases, and pointed out in the clinical histories that these factors seemed to be rather concurrent events than exciting causes, although to those who did not actually see them this statement may seem far-fetched. When, however, we come to the problem of cardiac infection and anæmia gravis we may find sometimes the utmost difficulty in making a differential diagnosis. There are many clinical features in common—such, for example, as the fever, the pallor, the long course, the rheumatic pains, the enlarged spleen and the liver, the breathlessness and purpura.

Cardiac infections and grave anæmia.—We are accordingly led here to comment upon an interesting point in the study of the grave anæmias in childhood dependent upon the behaviour of the heart sounds, which, in our experience, may be misleading even to careful observers. As the anæmia progresses the first sound at the impulse becomes short and abrupt,

and if in addition it is slightly reduplicated, the simulation of mitral stenosis is very close. We have not detected in such cases a definite thrill, but there is invariably a soft systolic murmur after the abrupt first sound. When the degree of anæmia is very profound a *diastolic* murmur may also become evident at the base, with its maximum usually just to the left of the sternum in the second intercostal space. When in addition there are fever, purpura and enlargement of the spleen, the resemblance to a progressive endocarditis is so close as to give rise to the greatest difficulty in the diagnosis. It is equally remarkable when there is improvement in the anæmia how completely these cardiac signs alter and return to the normal.

The following case is a striking example of this difficulty which we were unable to solve satisfactorily even after prolonged and careful study.

A girl, aged 11 years, was admitted to hospital on April the 5th, 1921, for anæmia and headache, pains in the limbs, and breathlessness of four weeks' duration. In the last five days she had become a peculiar yellowish colour. There was a history of chorea some years previously. Her mother had suffered from rheumatic fever three years previously and all the three children (girls) had suffered from chorea. They lived in a Welsh valley and in a damp, insanitary house, and the parents were first cousins. This child had had measles, chickenpox, and scarlet fever.

The pallor was striking, and with the rheumatic family history suggested at first glance either a virulent carditis or a malignant endocarditis of rheumatic or septic origin.

Examination showed an enlarged heart with loud, apical and basal systolic murmurs and a first sound so short and abrupt as to suggest a mitral stenosis. Pulse 140 to the minute. The liver was 3 in. below the costal margin and the spleen was also much enlarged. The fingers were clubbed a slight degree. There were no Osler's spots. The temperature was irregular, varying between 100·2° F. and 102·5° F.

There were no hæmorrhages. It was clear that on the diagnosis of malignant endocarditis all the symptoms including the enlarged liver and spleen could be explained, and that the abrupt first sound was not due to mitral stenosis but to the cardiac anæmia—these symptoms could be equally explained by an anæmia gravis. The first blood count was as follows: Red cells, 1,960,000; hæmoglobin, 25 per cent.; colour index, ·6; normoblasts, 2 per cent.; white cells, 16,400; polymorphonuclears, 75 per cent.; lymphocytes (small), 17 per cent.; large lymphocytes, 7 per cent.; large mononuclears, 1 per cent.; blood culture sterile. The results of the blood examination favoured a secondary anæmia.

There were no nodules, or arthritis or other evidence of simple acute rheumatism; no enlargement of the glands. The mouth was dirty, the tongue coated, and breath foul. There were no respiratory or nervous symptoms. The retina showed numerous hæmorrhages. The urine was free from blood and albumin. There was great doubt expressed as to the cardiac condition—some thinking there was mitral stenosis—others that all the cardiac sounds could be explained by the profound anæmia.

The course of the illness was a remarkable one. The temperature fell gradually, reaching normal seventeen days after admission. Anti-rheumatic vaccine was commenced on the 11th and persisted with through the illness. Cough, harsh breathing and crepitations developed at the left base, but there was no hæmoptysis. At the end of the month there was a definite improvement, the temperature was normal, the spleen smaller, and the red cell count had risen to 3,160,000 per c.mm., the colour index to ·9, the hæmoglobin to 55 per cent. The condition of the heart had improved, and the belief that there was organic valvular disease was shaken.

On May the 9th, seventeen days later, the lymphatic glands on the left side of the neck began to enlarge and became tender, and the temperature rose to 102° F. A severe tonsillitis developed rapidly. The spleen enlarged, there was præcordial pain, and a systolic and diastolic aortic murmur were heard. On the legs, in the popliteal spaces, buttocks and nose, small blebs developed which on rupture left small raw areas.

On May the 15th there was severe abdominal pain, rapidly followed by profuse hæmorrhage from the bowel, and tubular breathing was detected at the left base. The diagnosis now swung round strongly in favour of malignant endocarditis, and the outlook appeared to be hopeless. The right kidney now enlarged and became tender, but no blood or albumin appeared in the urine. After the hæmorrhage the spleen became smaller, receding from the level of the umbilicus. Purpuric spots appeared on the knuckles later, and with a reappearance of signs of mitral stenosis, a cantering cardiac rhythm, and the aortic murmurs the picture of an active endocarditis appeared to be complete.

Gradually an improvement set in, the temperature slowly falling, the spleen becoming smaller and firmer, and the child, now emaciated, began to feel much better and to gain ground though her colour was definitely icteric. This improvement was steadily maintained, although the spleen was noticed to vary in size from week to week. In August, three months later, no bruits were detected, and the diagnosis of endocarditis seemed to be very unlikely. In September she was stronger and the heart appeared to be normal in size and as to the sounds, but the spleen was palpable three fingers' breadths below the costal arch. The final blood picture was as follows: Red cells, 4,360,000; hæmoglobin, 60 per cent.; colour index, ·7; white cells, 14,800; polymorphs, 63 per cent.; small lymphocytes, 28 per cent.; large lymphocytes, 6 per cent.; large mononuclears, 1 per cent.; eosinophils, 1 per cent.; basophils, 1 per cent.

The patient left hospital in September with the diagnosis unsettled.

Von Jaksch's Anæmia Pseudoleukæmica Infantum.

In our list there are four cases which are included under this heading. The criteria which we have adopted are that the child should be under the age of four years, that the anæmia should be severe, that there should be an unusual proportion of myelocytes present in the peripheral blood, and that the spleen should be of considerable size. In addition, though not invariably present, normoblasts and megaloblasts are commonly found in the films of the blood.

Judged by these standards three of the four cases undoubtedly belong to the category of von Jaksch's disease, but as to the fourth (No. 1 in the list) there is room for hesitation. In this patient the hæmoglobin was certainly unusually high, and the proportion of myelocytes unusually low, nor had the spleen such a size as is usually associated with the disease. Moreover the erythrocyte count was higher than we should have expected and the improvement in the child's condition was exceptionally rapid. Still, the general appearance of the patient, coupled with the appearance in the blood of myelocytes, normoblasts and megaloblasts, incline us to believe that this is an example either of a comparatively late stage in convalescence from the disorder, or possibly an example of an exceptionally mild degree of the affection. But we must not fail to admit that such a case affords some support to the arguments of those who, unlike

ourselves, believe that von Jaksch's disease is not a clinical entity, but an unusually severe stage of any infantile anæmia, the uncommon features being merely an expression of the severity of the intoxication. If such a view be accepted the case represents a transitional stage between an ordinary " chlorotic " anæmia, and the severe grade of anæmia known by von Jaksch's name. We have in the first part of this paper given our reasons for dissent from this hypothesis and continue to hold that von Jaksch's disease is a separate clinical entity distinct from other forms of anæmia.

Of the other three cases there is nothing especial to note. Two recovered and one died. The post-mortem examination in this case revealed the usual conditions : petechiæ in many organs, an old blood-clot in the pelvis of one kindey, general moderate enlargement of the lymphatic glands and hypertrophy of the lymphoid tissue throughout the body, and a large firm spleen with marked fibrosis.

In all three cases myelocytes were present to the numbers of 1200–2000 per c.mm., and normoblasts and megaloblasts in varying proportions ; the hæmoglobin was 30 per cent. or less ; the red cells from 2,000,000 to 3,500,000 per c.mm., and in the acuter stages of the disorder a marked leucocytosis with a preponderating increase in the normal lymphocyte of the blood. Together with the great size of the spleen and the chronic course of the illness these patients appear to us to be properly classed as examples of von Jaksch's disease.

An Example of von Jaksch's Anæmia.

D. A—, female, aged 8 months. Was admitted under Dr. Hutchison on June the 12th, 1920. The infant had been wasting since two weeks of age, and had been fed for three weeks on cow's milk and barley water, six weeks on Nestlé's milk and then on Ideal milk. There had been repeated attacks of diarrhœa and vomiting. The child had congenital club feet. There were no teeth and there was some beading of the ribs. The abdomen was very large. The spleen extended three fingers' breadths below the costal arch, the liver one finger's breadth. There were some enlarged glands in both axillæ. Beyond these features there was nothing of note except pallor.

The blood count was as follows : Red blood cells, 3,930,000 per c.mm. ; hæmoglobin 32 per cent. ; colour index, ·4 ; normoblasts, 5 per cent. ; megaloblasts, 2 per cent. ; there were poikilocytosis and polychromatophilia. White cells, 15,200 per c.mm. ; polymorphonuclears, 36 per cent. ; small lymphocytes, 33 per cent. ; large lymphocytes, 16 per cent. ; large hyalines, 3 per cent. ; eosinophils, 1 per cent. ; transitionals, 1 per cent. ; neutrophil myelocytes, 9 per cent. ; eosinophil myelocytes, 1 per cent.

The child stayed in hospital until 1921 and improved. The spleen became smaller and in the last count there were no myelocytes.

A NOTE ON THE QUESTION OF THE CAUSATION OF POSTURAL OR ORTHOSTATIC ALBUMINURIA.

By F. Parkes Weber, M.A., M.D., F.R.C.P.

In the discussion on the after-history of cases of albuminuria occurring in adolescents, which took place on May the 2nd, 1911, at the Medical Section of the Royal Society of Medicine, I made some remarks on the favourable prognosis in such cases, and said that I did "not think that albuminuria occurring in apparently healthy adolescents could justly be regarded as belonging to any other class than orthostatic albuminuria, unless it could be definitely ascertained that the urine passed immediately on getting out of bed after the night's rest contained albumin." In a paper in the British Journal of Children's Diseases in 1911,* I referred to individual cases that I had met with, and since then I have not heard of a bad result, due to the albuminuria, in any of the cases in question, nor in any cases that I have met with since then. If anything, I am now still more convinced that in uncomplicated cases of orthostatic albuminuria the life may be regarded as a normal one from the life assurance point of view.†

In some way or other true orthostatic albuminuria is often connected with the tall "lanky" build of body suggestive of visceroptosis—the so-called "morbus asthenicus" or "asthenic constitution"—in which there is often some degree of lordosis likewise present. Hideo Saito (Japan), in his "Clinical Investigations on Orthostatic Albuminuria" in 1921,‡ concludes that orthostatic albuminurics have an asthenic constitution, that about half of them show lordosis, and that a large majority of them are in a state of vagotonia.

Jehle was the first to draw attention (1908 and 1909) to lordosis as an actual exciting cause, and as a result of his writings on the subject many papers were published under the heading "lordotic albuminuria." It is certain that in cases of orthostatic albuminuria temporary albuminuria can sometimes be produced by *artificial* lordosis, even when the patient remains lying in bed. The suggested explanation has been that in cases of lordotic albuminuria the albumin comes from the left kidney only and has to do with lordotic pressure on the left renal vein, which has to pass in front of the vertebral column and the aorta.

* F. Parkes Weber, "Remarks on Orthostatic Albuminuria," 'Brit. Journ. Child. Dis.,' 1911, viii, pp. 385–390.

† F. Parkes Weber, "Albuminuria in Relation to Life Assurance," 'Brit. Med. Journ.,' 1921, i, pp. 78–80.

‡ Hideo Saito, 'Am. Journ. Dis. Child.,' 1921, xxii, pp. 388–401.

Carl Sonne, of Copenhagen, in his paper, published in 1920,* on the ætiology of orthostatic albuminuria, arrived at that conclusion from his observations on six (five females and one male) typical cases of orthostatic albuminuria between the ages of 13 and 17 years. In these cases he catheterised both ureters and found that the albumin actually came only from the left kidney. Sometimes, according to Sonne, temporary anuria may be produced by catheterisation of the left ureter, as was known to Jehle. Sonne was able also to confirm the findings of Jehle and others, according to whom both granular and hyaline tube-casts can often be detected in the albuminous urine from cases of orthostatic albuminuria.

All this fits in with the well-known fact that in cases of chronic passive congestion of the kidneys, from various causes, the urine often contains albumin and few or many tube-casts; and in animal experiments albuminuria has been artificially produced by the circulatory disturbance connected with ligature of a renal vein or artery (Ludwig, Senator, etc.).

A remarkable case has been recorded by K. Vorpahl † of orthostatic albuminuria in which the albumin was proved by ureteric catheterisation to come from the right kidney. At first sight this case seems to be directly opposed to the above-mentioned findings of Sonne, who in his cases showed that the albumin came from the left kidney. Vorpahl's patient, however, who was a girl, aged 12 years, had spinal scoliosis, but hardly any appreciable lordosis, and it was suggested that the right renal vein and artery were pulled on owing to their being on the concave side of the lumbar scoliotic bend. The albuminuria occurred when the child was standing and when she was sitting up, and it was suggested that both these positions, in some way or other, intensified the drag on the right renal vessels, whilst the recumbent position lessened it.

W. Rieser and S. L. Rieser, of New York, in their recent paper on the "Ætiology of Orthostatic Albuminuria" (1922) ‡ confirm the mechanical theory, namely, that the albuminuria is due to passive venous congestion of the left kidney. They conclude that in such cases the left renal vein is compressed between the abdominal aorta and the mesenteric artery, when the aorta is pushed forward as a result of lordosis or when the mesenteric artery is stretched by ptosis of the mesocolon.

* Carl Sonne, "Beitrag zur Aetiologie der lordotischen (orthostatischen) Albuminurie," 'Zeitschr. für klin. Med.,' 1920, xc, pp. 1–6.

† K. Vorpahl, "Ueber einseitige orthostatische Albuminurie," 'Berl. klin. Woch.,' 1910, xlvii, p. 827.

‡ W. Rieser and S. L. Rieser, 'Journ. Amer. Med. Assoc.,' 1922, lxxviii, pp. 644–647.

Nevertheless, it is clear that lordosis cannot be the only factor in the production of orthostatic albuminuria, even in cases in which ureteric catheterisation proves the albumin in the urine to come only from the left kidney. There must be an individual predisposition of some kind. It is often in the morning that the albuminuria is most marked, even though the orthostatic position is maintained at other times of the day. Besides this cyclic character of the albuminuria, the age-incidence has to be remembered, though it is a mistake to think that orthostatic albuminuria is altogether confined to childhood, puberty, and very early adult life. It may be met with up to twenty-five to thirty years of age and possibly even later.* D. C. Parmenter† quotes T. B. Barringer‡ as stating that ten to eleven years after the examination of some seventy men who originally had albuminuria, 40 per cent. still showed it, though none of these 40 per cent. had nephritis or symptoms thereof. I do not, however, know in how many of Barringer's cases the albuminuria was of the true orthostatic type.

Moreover, lordosis does not always produce orthostatic albuminuria, and this fits in with the fact that chronic passive congestion of the kidneys in heart disease, etc., is not in some cases (for a long time at least) accompanied by albuminuria.

In this connection I should like to mention, for what it is worth, that in two young men with extreme cyanosis from congenital heart disease I found that orthostatic albuminuria was present. The first case was that of a man, aged 22 years§; the age in the second case was 29 years.‖ These are the only cases of congenital heart disease (probably pulmonary stenosis) in patients who have survived childhood in which I have been able to examine the urine for orthostatic albuminuria. But F. Eigenberger¶ refers to the case of a young man with polycythæmia rubra (secondary to cardiac disease, if I rightly understand him) in whom the presence of orthostatic albuminuria was discovered; a lordotic position of his body caused his urine to contain albumin, a little blood and abundant cylindroids. Orthostatic albuminuria was likewise found in a young man, aged 18 years, whose case, described by G. Herrnheiser,**

* *Cf.* F. Parkes Weber, 'Brit. Med. Journ.,' *loc. cit.*

† D. C. Parmenter, "Observations on the Significance of Functional Albuminuria in Young Men at Harvard University," 'Boston Med. and Surg. Journ.,' 1920, clxxxiii, pp. 677–681.

‡ T. G. Barringer, "Prognosis of Albuminuria," 'Arch. Int. Med.,' 1912, ix, p. 657.

§ F. Parkes Weber, "Congenital Heart Disease, with Extreme Secondary Polycythæmia and Orthostatic Albuminuria," 'Edinburgh Med. Journ.,' 1909, New Series, ii, p. 18.

‖ Weber and Dorner, "Congenital Pulmonary Stenosis," 'Proc. Roy. Soc. Med.,' Clin. Sect., 1911, iv, p. 85.

¶ F. Eigenberger, 'Zentralbl. f. inn. Med.,' 1920, xli, p. 354.

** G. Herrnheiser, 'Deut. Arch. f. klin. Med.,' 1919, cxxx, pp. 315–330. There was no splenomegaly. The brachial systolic blood-pressure was 110 mm. Hg.

was, I suppose, one of secondary polycythæmia rubra due to some kind of chronic circulatory disturbance.

The arguments in favour of the mechanical factor in orthostatic albuminuria are supported by certain cases of so-called *clinostatic albuminuria*, in which apparently some mechanical factor, such as the pressure of an enlarged spleen, gives rise to albuminuria in the recumbent but not in the standing position. Sir H. D. Rolleston* observed this kind of albuminuria in cases of leukæmia, hepatic cirrhosis and splenic anæmia and referred to a similar observation by H. Falkenheim† in a man, aged 50 years, with hepatic cirrhosis and an enlarged spleen. Dufour and Muller‡ describe another kind of "clinostatic albuminuria" in a boy, aged 16 years, which they attribute to nervous exhaustion from masturbation. J. A. Amblard§ has recorded "nocturnal clinostatic albuminuria" in gouty subjects, and M. W. Thewlis‖ thinks that prolonged lying in bed may give rise to the presence of tube-casts in the urine of aged patients.

In regard to the possible occurrence of hæmaturia in cases of orthostatic albuminuria, it seems to me that, when a marked lordotic position mechanically disturbs the circulation in the left kidney, the artificial fixation (for some orthopædic reason) of the lordotic position by an orthopædic plaster-jacket—so that the lordosis remains, *even in the recumbent position*, until the plaster-jacket is removed—might aggravate the renal disturbance so as to cause hæmaturia also. This is confirmed by a case of which I have heard through the kindness of Sir H. D. Rolleston and Dr. G. C. Willcocks (of Sydney, Australia), but I think that the case has not yet been published.

* H. D. Rolleston, 'Trans. Med. Soc. Lond.,' 1902, xxv, p. 196. Similar observations by Dauchey are referred to by P. Mougene de Saint-Avid, in a Paris medical thesis (1912, p. 106) on "Les albuminuries intermittentes irrégulières," but I have not been able to find Dauchey's work.

† H. Falkenheim, "Regelmässig intermittirende Albuminurie," 'Deut. Arch. f. klin. Med.,' 1884, xxxv, p. 446.

‡ Dufour and Muller, "Sur une singulière albuminurie clinostatique," 'Bull. de la Soc. de Péd. de Paris,' 1913, xv, p. 271.

§ J. A. Amblard, 'Gaz. des. Hôp.,' 1911, lxxxiv, p. 634.

‖ M. W. Thewlis, 'Geriatrics,' London, 1919, p. 52.

A CASE OF ANEURYSM OF THE DUCTUS ARTERIOSUS.*

By Robert Hutchison, M.D., F.R.C.P.

The patient, a girl, aged 6 years, was admitted to the London Hospital on March the 4th, 1922, and died twelve days later from a sudden and profuse hæmoptysis. There was a history of measles followed by diphtheria in the preceding October, and six weeks later she suffered from "pleurisy" and had not been well since. On admission she was complaining of pain in the left side of the chest with cough and feverishness.

On examination she was a pale, thin child, crying intermittently with pain in the chest. No cyanosis or finger-clubbing.

The left side of the chest was somewhat flattened, the apex-beat was diffuse and felt in the fifth and sixth spaces just outside the nipple line. There was a soft systolic murmur at the apex and over the sternum. Examination of the lungs showed some dulness in the left infra-clavicular and suprascapular regions with diminution of the breath-sounds and some crepitation over the right apex in front. Examination of the other organs revealed nothing abnormal.

As the case progressed the dulness at the left apex became more pronounced and the apex-beat of the heart extended further to the left. The short systolic murmur disappeared. The temperature remained moderately elevated (100°–101°F.) throughout. On March the 15th there was a small hæmoptysis, and the next day a very profuse one which resulted in almost immediate death.

Post-mortem Report by Dr. H. M. Turnbull.

Hæmoptysis; broncho-pneumonia and empyema; rupture of aneurysm of ductus arteriosus into lung.

False aneurysm (7 cm. diam.) beneath pericardium in front of and to left side of roots of great vessels. Wall of aneurysm—up to 0·4 cm. thick. Lining—composed of trabeculated organised clot and the contents of a nodule of dense laminated clot.

Aortic orifice of ductus arteriosus (0·7 × 0·4 cm.) opening into a true aneurysmal dilatation of aortic portion of ductus arteriosus; sharply cut free edges of true aneurysm project into false aneurysm.

Pulmonary end of ductus arteriosus closed. Great thinning of wall of pulmonary artery at bifurcation where adherent to lower anterior border of false aneurysm. This thin area (1·5 × 1 cm.) covered by layer of thrombus.

Aneurysm adherent to upper lobe of left lung and eroding into it to a depth of 1 cm. Organised fibrous obliterating pericarditis. Adhesions most dense over left auricle.

* The specimen from this case was shown at a meeting of the Section for the Study of Disease in Children of the Royal Society of Medicine on March the 24th, 1922.

Hypertrophy of heart, greatest on right. , Compression of pulmonary stem and of ascending aorta and arch. Dilatation of ascending aorta (4·3 cm. circumference) and of descending thoracic aorta (2·8 cm. circumference).

Lungs: Left: Upper lobe—œdema and hæmorrhage consolidating upper and anterior fourth, around the perforation of the aneurysm. Emphysema of rest of left lung, except for small patch of collapse at lower border. Right: At anterior border of middle lobe a small area of chronic inspissated purulent pneumonia. Small empyema, partly inspissated, continuous with this, lying between anterior border of right upper and middle lobes and chest-wall (size 4·5 × 3 cm.). Obliterating fibrous pleural adhesions flecked with inspissated pus.

Blood in bronchi and trachea.

Stomach: Mucous catarrh. Blood-clot 2 oz.

Spleen: Red, chronic septic spleen.

Liver: Severe parenchymatous degeneration.

Kidneys: Ditto.

Congenital abnormality (double pelvis and double ureter uniting immediately above bladder-wall) on right side.

Remarks.

The case is a good example of aneurysmal dilatation of a persistent ductus arteriosus of which several have been described.(1) The fact that the pulmonary end of the ductus was closed no doubt accounted for the absence of the characteristic murmur. It is unfortunate that a radiograph of the chest in this case was a failure as otherwise it might have revealed what is diagnostic of the condition—a pulsating projection like a cap on the top of the heart shadow. Whether the development of the aneurysm was caused by inflammatory disease of the wall of the ductus supervening upon the infection of the lungs I am unable to say.

Reference.

(1) 'Pfaundler and Schlossmann's Diseases of Children' (translation by Shaw and La Fétra), iii, p. 481.

PURPURA FULMINANS FOLLOWING MEASLES.*

By E. H. Kelly, M.B., D.P.H., B.Sc.,

Assistant Medical Officer, Grove Fever Hospital, London.

Although fifty cases of purpura hæmorrhagica and purpura fulminans have been reported after scarlet fever during the last twenty years (McConnell and Weaver), only a few cases have been described following measles. Two of Elliott's collection of fifty-six cases (including cases of purpura hæmorrhagica) and one of Little's cases followed an attack of measles.

It is of local interest that four previous cases of purpura fulminans

* The case was reported at the Section for the Study of Disease in Children of the Royal Society of Medicine on April the 28th, 1922.

have been reported from this hospital within the last twenty years, three after scarlet fever by Biss, Rolleston and McCririck, and McCririck and one after diphtheria by Gunson. The fifth case, the subject of the present note, was a male infant, aged $1\frac{5}{12}$ years, who was admitted to the Grove Fever Hospital, certified measles, broncho-pneumonia and cancrum oris. On admission he showed a fading macular erythema on trunk and limbs, conjunctivitis, coryza. The upper lip showed marked induration and swelling extending into the inner half of the left cheek. Both lips were fissured and covered with crusts. The buccal mucous membrane and the fauces were injected, but clean. The temperature was 102° F., pulse 124, and respirations 56. Moist râles with coarse rhonchi were heard at both bases of the lungs. The other systems showed nothing abnormal. 30 c.c. of antistreptococcal (polyvalent) serum were injected into the abdomen.

The child's condition improved daily, and on the fourth day after admission the temperature was normal and the swelling of the lip and cheek had subsided. On the twelfth day he developed his serum rash—a blotchy erythema over trunk and limbs. This faded the following day. On the fifteenth day his temperature rose to 103°F., his pulse was rapid and small, and his respirations 50. He vomited several times that day and complained of abdominal pain. The examination of the abdomen showed nothing abnormal. He remained very ill the following day, and in the early morning of the next day (seventeenth day after admission) he developed small petechiæ over the trunk and limbs and a symmetrical dark purpuric area shaped like a butterfly's wings, occupying the cheeks and nose. The temperature was 102° F., pulse rapid, and respirations shallow. The child died in the afternoon of that day, seventeen hours after the first appearance of the purpura.

The child had been healthy previous to this attack of measles. The family history was not of interest.

Permission was not given for a post-mortem examination and a blood-count was not made before death.

Henoch, in 1886, gave the name of purpura fulminans to a condition characterised by rapid development, great prostration, often resulting in death within twenty-four hours, and showing hæmorrhages limited to the skin, but complete absence of hæmorrhages from the mucous membranes, and absence of hæmorrhages in the organs after death, under the name "purpura fulminans."

The ætiology is still unsettled. Elliott collected reports of fifty-six cases. These included also cases of purpura hæmorrhagica. Of these eleven followed scarlet fever and two followed measles. McCririck, in 1912, in discussing his case, stated that of sixty-four cases of purpura

fulminans published up to date, seventeen had followed scarlet fever, occurring usually in the second, third or fourth week of the disease.

H. D. Rolleston and Molony describe eleven cases of purpura developing as a terminal phase in fatal cases of epidemic diarrhœa in children under one year. In one case *B. enteritidis sporogenes* was isolated in pure culture from the heart's blood.

Hanot and Luzet obtained the meningococcus in pure culture in exudation on the meninges in a case of cerebrospinal meningitis with purpura, also in the liver, spleen, and uterus. The fœtus showed hæmorrhagic patches on the serous membranes with streptococci in the liver and heart.

Netter and Mozer found purpura in about 30 per cent. of their cases of cerebrospinal meningitis, and Herrick thinks that rapidly fatal cases of purpura are secondary to meningococcal infection. Graham Little describes eleven cases (one following measles) associated with suprarenal hæmorrhages. He pointed out that the medullary portion of the gland was most affected, giving the appearance of hæmorrhagic infarcts. He

suggests this as an explanation of the sudden prostration and death in previously healthy children.

It has been suggested that the purpura and severe general disturbance in the case reported were manifestations of serum sickness following the injection of horse-serum. As cases of purpura fulminans were described before the use of horse-serum and as a large proportion of the cases have followed scarlet fever in which no serum was used, the injection of serum was probably not the cause.

I have to thank Dr. J. H. Whitaker, Medical Superintendent of the Grove Hospital, for permission to publish this case; Dr. J. D. Rolleston for much help with regard to the literature on the subject; and Dr. T. Brushfield, of the Fountain Mental Hospital, who took the photograph shown.

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Royal Society of Medicine.

CLINICAL SECTION.

October the 14th, 1921.

Case of Renal Dwarfism with Optic Atrophy.—Dr. A. Feiling and Dr. W. L. Holyoak showed a boy, aged 12½ years, who had come under observation for defective vision in the left eye. He was said to have had whooping-cough at the age of 4, after which the sight became worse and the legs became crooked. Present condition: Height 43½ in., weight 3 st. 13 lb. 8 oz. Mental state quite normal. Left eye-vision reduced to perception of large moving objects only; primary optic atrophy in left eye. Right optic disc pale, but vision $\frac{6}{9}$ and $\frac{6}{6}$ with correction. Visual field in right eye full.

Very marked degree of genu valgum in both legs. Urine: The amount had varied from 26 to 40 oz. in twenty-four hours. Specific gravity 1012, acid, clear; constant albuminuria. Amount of albumin 0·25 per cent. or 1·1 gr. per ounce. A few hyaline and granular casts found. No definite cardio-vascular changes; blood-pressure 95 mm. Hg. systolic and 70 diastolic. Wassermann reaction in the blood negative. Cerebro-spinal fluid: Wassermann reaction negative; cell count 2 to 3 per cubic millimetre; no globulin. On X-ray examination the sella turcica was small and ill-developed, but otherwise no definite abnormality could be detected.

November the 11th, 1921.

Case of Cleido-cranio Dysostosis.—Mr. C. E. Shattock showed a girl, aged 6 years and 8 months. No history of the condition being hereditary. Right shoulder held lower than left. Shoulders could be brought together anteriorly. Right clavicle showed an absence of bone in its middle third, where it was represented by a fibrous band. Left clavicle shorter than normal, appeared to be bony, and was loosely articulated with sternum and acromion. Toes stunted. Nails of fingers and toes short. X-ray examination: Skull: Thinning of bone in region of bregma, stunted development of nasal bones, absence of frontal sinuses. Clavicles: Imperfect ossification, more marked in right clavicle, where there was absence of bone in middle third. Toes: Absence of epiphysial centres in phalanges; abnormal breadth of phalanges of hallux; poor development of ungual tuberosities. Fingers: Irregular epiphysial lines in some of the intermediate and terminal phalanges; proximal and distal epiphysial centres in second and fifth metacarpal; poor development of ungual tuberosities.

January the 13th, 1922.

Case of Plexiform Neuroma.—Mr. Philip Turner showed a girl, aged 14 years, with a diffuse tumour involving the left external ear, parotid region of the face and occipital region of the scalp. The mother said that the swelling of the external ear had been present since birth, but that during the six months before her admission to hospital in May, 1921, it had increased in size, so that it completely obstructed the external auditory meatus, rendering the child deaf on that side unless she retracted and raised the pinna. Extension of the growth to the parotid and occipital regions had been noted for the last eighteen months. The tumour was soft and compressible, but did not fluctuate; it appeared to have caused absorption or displacement of the cartilage of the external ear. There was a well-marked constriction between the swelling of the ear and that in the occipital region, which extended upwards to the vertex of the skull, but the tumour was continuous from the left parotid region to the occipital region beyond the mid-line, while its vertical extent was from the upper half of the neck to the vertex. Its outline was ill-defined, but several hard nodules could be felt in the mastoid region and near the external occipital protuberance. Recently there had been a remarkable growth of hair in front of the ear. There was but little pain, though the hard nodules were tender when pressed. The nerves involved appeared to be the ascending superficial branches of the cervical plexus. In May, 1921, portions of the tumour were removed from behind the ear, in the hope that the obstruction of the external audi-

tory meatus might be removed. This was to a certain extent accomplished, as she was now able to hear with the left ear. Though the extent of the tumour had increased but slightly, new tissue appeared to have formed in the region of the old operation. Histological examination showed that the growth was a plexiform neuroma.

Hodgkin's Disease with Long History and Absence of Constitutional Symptoms.—Mr. W. H. Ogilvie.—The patient was a boy, aged 13 years. In July, 1918, his mother first noticed small swellings in the left side of the neck, which were diagnosed as tuberculous glands, and treated by local and constitutional measures for four months. In December, 1918, a chain of glands was removed from beneath the left sterno-mastoid. Section of one of the glands showed typical lymphadenoma. Shortly after his discharge the swellings reappeared, and he was treated from January, 1919, to February, 1920, by X rays. As the swellings continued to increase, the treatment was discontinued. In November, 1920, as the swelling, after two years, was still limited to the left side of the neck and as his general health was excellent, he was readmitted to hospital for further surgical treatment, and glands were removed on November the 29th and January the 23rd, 1921. The swellings reappeared, and he was again given X-ray treatment from February to April, 1921. Present condition: Operation scars on left side of neck. Group of enlarged glands in submaxillary region and over masseter, also some obstructive lymphatic œdema of left side of face. On the right side a mass of glands extended from the clavicle to the mastoid process, mostly behind the sterno-mastoid. There was also a mass in the left axilla. The right axilla and groins were free. Rounded lower border of liver could be felt $1\frac{1}{2}$ in. below costal margin, and spleen also descended $\frac{1}{2}$ in. below tenth costal cartilage. Chief complaint was pain in left axilla. Temperature normal during three weeks' observation. Pulse varied from 60 to 100. Blood: red cells, 4,890,000; white cells, 11,000; hæmoglobin, 75 per cent.; colour index 0·7. Blood-pressure 110 mm. Hg.; nervous system and urine normal. Mr. Ogilvie now proposed to treat him with radium.

February the 10th, 1922.

Syphilitic Ulcer of the Tongue in a Child.—Mr. W. E. Tanner.—The patient was a boy, aged 4 years, who, on December the 13th, 1921, complained of pain in the tongue. The mother found a deep fissured ulcer at the tip and thought that he had bitten his tongue. There were two younger children, both stated to be healthy. The last child was born two weeks ago; one miscarriage. Last summer the boy had an affection of the eyes for which he attended hospital every day for two months. The father had a positive Wassermann reaction, but the mother refused to have a blood-test.

The parents had no evidence of active syphilis at the present time. The child was well grown and of healthy appearance, with no sign of congenital syphilis except that the head was large and square. On January the 13th there was a large ulcer, which had destroyed the tip of the tongue, spreading on to the frænum. The edges of the ulcer were sharply defined and the base was covered with a slough. The submaxillary glands on both sides of the neck were enlarged.

Decayed teeth had been extracted. There was no sign of secondary syphilis. The Wassermann reaction was positive. The appearance of the ulcer suggested a gumma; the glands did not appear to be so enlarged and

hard as with a primary sore. On January the 23rd an injection of 0·18 cg. of sulfarsenol was given intramuscularly into the buttock, and mercury and iodide were given by the mouth. Within a week the ulcer was quite clean, with healthy granulations and commencing epithelialisation. The glands were small but shotty. Three injections of sulfarsenol had now been given at intervals of a week. A piece of the ulcer removed for microscopical examination had been reported to be gummatous by three pathologists.

SECTION OF DERMATOLOGY.

December the 15th, 1921.

Case of Multiple Nævi.—Dr. A. M. H. Gray showed an infant, aged 5 weeks, which had been born without any skin lesion. On the second day of life a little spot came out on the left eyebrow, and since then lesions had been coming out every day, and were still appearing. Almost every part of the body was affected. They were now very numerous, some 50 or 60 in number. Some of them, especially the early ones, appeared to have the more or less typical characters of ordinary stellate nævi, whereas others were more like simple angiomata.

Nævo-xantho-endothelioma (?) with Epidermolysis Bullosa.—Dr. G. W. Sequeira showed a female infant, aged 17 months, with xanthoma-like lesions on hands and feet. They were first observed soon after birth and appeared in clusters on the nose and other parts. The mother stated that many of the clusters had disappeared. The lesions were now confined to the hands and feet. The infant had also suffered from epidermolysis bullosa since birth, any damage to the skin from trauma, such as blows, friction or pressure, being followed by characteristic bullæ. The child's mother had also suffered from a combination of the two diseases and still showed some nodules, and blisters formed after damage to the skin, although not so readily as they did when she was younger. The child's grandmother stated that she had had the same yellowish nodules when a child, and used to be subject to blisters when she injured her skin. Her father had also suffered in the same way after injuries, as had also her sister and her sister's two sons. One of these boys had died from diabetes at the age of 12. The nails of one or two of the fingers showed desquamative changes, probably the result of blebs formed beneath and around the nail, leading to changes in the nail plate and thus interfering with its growth.

January the 19th, 1922.

Case of Lichen Obtusus Corneus.—Dr. W. Knowsley Sibley showed a girl, aged 11 years, who had had for two years a papular eruption over the body. The mother said that the lesions commenced on the legs and had gradually spread over the body. At present the shoulders were extensively affected; she had lesions all down the arms, a few lesions on the front of the chest, and the legs were much involved. She said that they did not itch, but she had picked off the heads of a large number of the papules, and the mother said that the girl scratched herself during sleep. Vesicles had not been seen. She had not been given any treatment internally, but she

had had three small applications of X rays to the shoulders, and there was a considerable subsidence of the lesions in these regions. She had typical lichen planus papules over the elbows and shoulders, and many of the lesions above described were horny in character, especially those on the anterior aspect of the legs and shoulders. Dr. Sibley suggested that they were allied to those found in lichen obtusus corneus. Some of the lesions were distinctly annular, with recent small lichen planus papules occurring in the periphery of the lesions. A few of the lesions, especially about the elbows, were in a linear formation.

Parakeratosis Variegata.—Dr. H. W. Barber showed a boy, aged 13 years, who had come to him a few weeks ago with an eruption which he had diagnosed as belonging to the parapsoriasis or parakeratosis variegata group. The eruption had first appeared at the end of last September, first on the arms, then on the shoulders and legs. There were no subjective symptoms and very little scaliness. On the shoulders particularly there were telangiectatic spots. He proposed to treat the case with ultra-violet light.

February the 6th, 1922.

Case of Unilateral Morphœo-sclerodermia Faciei.—Dr. George Pernet showed a boy, aged 11 years, who had attended his out-patient department four months previously with a patch on the centre of the right cheek about the size of a florin, which, when first noticed two months before then, was about the size of a sixpence. There was also a certain amount of longitudinal morphœa occupying the lower right eyelid. The condition was improving under zinc ionisation.

Psoriasis in an Infant.—Dr. Haldin Davis showed an infant, aged 7 months. When 4 months old a scaly eruption developed; this gradually spread so that now it was almost universal. There were a few unattacked areas on the chin, hands and feet. The diagnosis was psoriasis in spite of the patient's age, though it had now become almost a case of dermatitis exfoliativa. A point in favour of the diagnosis was that the mother had had psoriasis and that the eruption when the child was first seen was circinate.

Lichenoid Linear Nævus.—Dr. E. G. Graham Little showed a male infant, aged 18 months, who had had the condition since very shortly after birth. At present there were two broad streaks consisting of raised red discrete lesions resembling lichen planus of a somewhat hypertrophic type, extending side by side and at a distance of half an inch or so from the buttock to the heel on the left side.

Case of Grouped Comedones.—Dr. Graham Little also showed a female child, aged 1 year, with a very extensive eruption of so-called grouped comedones, extending from nape to waist over the back and from neck to navel in front. The mother had rubbed the child with olive oil in which she had dissolved a block of camphor.

SECTION OF LARYNGOLOGY.

April the 1st, 1921.

Case of Lateral Proboscis.—Mr. Philip Franklin showed a male infant with absence of the nasal fossa and accessory sinuses on the right

side and a nasal proboscis or tube resembling a penis in form situated above and internal to the right eye. The central canal of the appendage could be traced to the depth of the orbital cavity. Movement of the appendage was quite apparent, and this suggested that the facio-nasal muscles were developed. The free extremity of the appendage, except for the perforation, showed the septum of the nasal vestibule which at times caused occlusion of the anterior nares. It was obvious that the appendage must be removed, and Prof. Keith had suggested that the eye should be removed at the same time in order to avoid the formation of a fistula and also subsequent lacrymation. A fistula was particularly to be avoided, since it might lead to meningeal infection. The appendage extended to the region of the cribriform plate and olfactory lobe.

Scarlatinal Scarring of the Pharynx.—Dr. Dan McKenzie showed a middle-aged woman who, having discovered a "lump" in her throat a few weeks ago, had come to hospital for advice. On examination it was seen that the lump was probably the uvula shifted over to the right side of the palate by cicatricial contraction. The scarring with its fine edges, thin substance and smooth surface was unlike an old tertiary lesion, and the Wassermann reaction was negative. The patient had had a severe attack of scarlet fever at the age of five years and the present condition was most probably due to scarlatinal angina.

December the 2nd, 1921.

Repair of Nasal Deformity caused by Syphilis.—Dr. Douglas Guthrie showed photographs of a girl, aged 18 years, who, between the ages of 9 and 16, had suffered from inherited syphilis, which had destroyed the cartilaginous part of the septum and the columella and caused an extreme degree of saddle-nose deformity. After three months of anti-syphilitic treatment the Wassermann reaction became negative and operation was undertaken. The nasal bridge was reconstructed by a costal cartilage graft, and a columella was fashioned from an upper flap from the skin of the vestibule and two lower flaps from the upper lip. Dr. Guthrie had operated upon seven such cases, two of them due to syphilis, five caused by injury. The traumatic cases were naturally the more favourable, but in all the results were good.

SECTION OF ODONTOLOGY.

January the 23rd, 1922.

Mandibular Sarcoma in an Infant.—Mr. Stanley Mummery.—The patient was a baby, aged 9 months, the daughter of a medical man. In February, 1921, a swelling developed on the left side of the mouth, and the local doctor who examined it, finding it was cystic in character, inserted a trocar and cannula, and drew off a quantity of serum. The swelling very soon re-formed, and the child was brought to Mr. Mummery on March the 18th, when the following condition was found: There was a large swelling in the left cheek, pressing up the lower eyelid, and filling the left side of the mouth nearly to the middle line. The swelling was tense and fluctuating, and appeared to spring from the posterior portion of the lower

left mandible. No œdema was present. A hard body resembling a tooth could be felt on the outer side of the tumour next the cheek. A skiagram showed the swelling to be cystic with a definite limiting wall; the secondary temporary molar was embedded in its outer wall and was raised half an inch above the mandible. A shadow which appeared to be the germ of the first permanent molar could just be distinguished below the swelling. On the strength of the clinical and X-ray evidence Mr. Mummery thought the growth was possibly a dentigerous cyst, and decided to operate next day. At the operation the mucous membrane was incised, disclosing a purple cyst wall underneath. This was gradually dissected out until the posterior part was reached, which was found to be firmly adherent. The tumour was then opened, and a quantity of blood-stained mucilaginous material and serum evacuated. Examination of portions of the cyst wall by Mr. Howard Mummery showed epithelial strands and traces of an epithelial lining in places, thus bearing out the clinical resemblance to a dentigerous cyst. The mucilaginous material examined by Dr. Eastes proved to be sarcoma of the small round-celled variety. Sir Arbuthnot Lane then decided to try radium, and a small tube was inserted into the wound for six hours. A week later the tumour had practically disappeared; beyond a slight fulness in the sulcus perceptible to the finger there was no sign of any swelling whatever. No further change took place for several weeks, but on June the 14th, nearly three months after the operation, the child was brought up with a recurrence of the swelling. The tumour was then of the same size and in the same position as formerly. On June the 16th a second operation was performed by Sir Arbuthnot Lane of a rather more extensive nature than the first. The contents of the growth were less fluid than before, and resembled transparent jelly. The rough exposed bone of the mandible could be felt at the bottom of the wound. Radium was again employed, and the tumour again shrank almost to vanishing point. On October the 22nd Mr. Mummery saw the patient again, as the lower temporary canine on that side had become loose and was giving trouble. The tumour had reappeared, but was of nothing like its former size. Mr. Mummery removed the canine, which was embedded in sarcomatous tissue, and inserted a small radium tube into the socket for a few hours. The child lived until January, 1922, by which time the sarcoma had made rapid progress, entirely filling the mouth and pressing on the fauces. Towards the end feeding became impossible, and partial asphyxia hastened the inevitably fatal termination.

SECTION OF OPHTHALMOLOGY.

December the 9th, 1921.

Lobulated White Mass at Macula.—Mr. C. J. Longworth Blair.—The patient was a boy, aged 11 years. Vision in the right eye had been defective for about the last two and a half years. He was stated to have had laryngitis, whooping-cough and croup during or about the years 1914–15, also an attack about 1914-15 of chorea. The point of special interest consisted in the appearance of the lobulated white mass at the macula.

Double Congenital Ptosis; Motais' Operation on Right Eye.—Mr. R. Affleck Greeves.—The patient was a male infant, aged 12 months,

with complete congenital ptosis of both eyes. Motais' operation was performed on the right eye on October the 19th, 1921. The left eye had not been dealt with.

January the 13th, 1922.

Growth on the Conjunctiva in an Infant.—Mr. P. G. Doyen showed a female infant, aged 4 months, with swellings to the outer side of the limbus in each eye noticed since birth. Other congenital defects were a bifid tongue and three phalanges on the right thumb. In the subsequent discussion the general opinion was that the growth on the conjunctiva was a dermoid.

Osteomyelitis of the Upper Jaw in Infants.—Mr. M. S. Mayou reported two cases. Case 1: An infant, aged 4 months, was admitted to hospital with a temperature of 100·8° F., pulse 102, respirations 28, with a sinus and swelling below and to the outer side of the right eye. Two weeks before admission the swelling, which was attributed to an insect-bite, appeared below the right eye. An incision had been made into it by the family practitioner parallel to the lower margin of the orbit and a quantity of pus evacuated. Two and a half weeks after admission the sinus was opened up and scraped. The patient remained in hospital for two months and was finally discharged with the sinus unhealed. A piece of bone was extruded from the sinus on December the 29th, 1921. Case 2 was an infant of about the same age admitted to hospital with a swelling below the right eye, the lids of which were red, œdematous and tender. The eyeball was proptosed. The child was ill, temperature 104° F. and pulse 140. Several days before admission an abscess in the hard palate had been excised by the general practitioner, but this had healed completely on admission. An incision was made parallel to the lower border of the orbit and carried down to the bone; this liberated a quantity of pus, and bare bone in the floor of the orbit could be felt. An immediate improvement took place in the child's condition, the temperature falling to normal in about a week. A sinus persisted until the child was discharged from hospital six weeks later, but had now healed. Mr. Mayou had seen the first case of this kind in 1901 in an infant of much the same age, when practically the whole of the upper jaw came away piecemeal as a sequestrum. The source of infection was difficult to understand as these children had no teeth. As a rule the nose could also be excluded, as there were practically no sinuses. Mr. Mayou thought that the infection was probably metastatic as in the cases of the long bones. The Wassermann reaction was negative in the first case, and in the second case the general practitioner said that there was no suspicion of syphilis either in the parents or in the child.

SECTION OF SURGERY: SUB-SECTION OF ORTHOPÆDICS.

April the 5th, 1921.

Case of Bilateral Radio-ulnar Synostosis associated with Bilateral Congenital Dislocation of Hips.—Mr. E. Laming Evans.—The patient was a girl, aged 5 years, who was first seen in May, 1918, when the mother stated that the child was unable to roll the forearms. There were three sisters and one brother, and no history of deformity in the family.

On examination synostoses of the upper end of the radius and ulna were found on both sides; the forearms were in a position of pronation. A bilateral congenital dislocation of the hips was also present, and was reduced in September, 1918. Mr. Laming Evans had not attempted to treat the radio-ulnar synostosis for the following reasons: (1) That the functional disability was slight and could largely be compensated by external rotation of the shoulder; (2) that the attempts at nearthrosis had resulted in the cases he had seen in failure.

Case of Multiple Cartilaginous Exostoses associated with Congenital Dislocation of the Hip.—Mr. Laming Evans also showed a girl, aged 9 years, first seen at the age of 2 years 9 months in October, 1914, when the left leg was observed to be shorter than the right. Examination showed dislocation of the left femur on to the dorsum ilii. Nothing abnormal detected either on femoral head or at site of acetabulum; thickened mass felt on anterior surface of left femur below level of great trochanter, also small irregular mass on crest of right ilium. There was no history of deformity in either father's or mother's family.

The case was not seen again until March, 1921, when it had developed into a typical example of multiple cartilaginous exostoses. Exostoses could be felt on the left femur, at the insertion of the adductor magnus, and at the upper end on the anterior surface just below the level of the great trochanter, on the left tibia at the insertion of the sartorius and at the lower end of the left fibula. The X-ray appearance of the left acetabulum suggested a cartilaginous exostosis arising from the Y-shaped cartilage, but this could not be confirmed by palpation. Exostoses were also present on the right iliac crest, right femur and right fibula, lower end of the right radius and vertebral borders of scapulæ. Mr. Evans considered that this was a case of congenital dislocation of the hip, in which the cause was a shallowing of the acetabular floor caused by a cartilaginous exostosis from the Y-shaped cartilage.

Société de Pédiatrie, Paris.

January the 17th, 1922.

Permanent Slow Pulse associated with Patent Interventricular Septum.—MM. Barbier, Lesné and Mouquier showed a boy, aged 15 years, who, apart from measles at two, had been in good health until six months previously, when he began to be short of breath on going upstairs or running. No other symptoms such as cyanosis or attacks of suffocation were present. On examination systolic pulsation was visible in the fourth and fifth left intercostal spaces, and a thrill was felt in the mesocardiac region. Along the margin of the sternum a systolic murmur was heard, most distinct at the inner end of the third intercostal space. The pulse was 44 a minute. The electro-cardiogram showed a complete auriculo-ventricular dissociation. Production of the oculo-cardiac reflex gave rise to a syncopal attack. The congenital nature of the affection appeared to be established by the presence of other developmental errors, such as costal nodules, high arched palate, asymmetrical skull, small testes, infantile voice and absence

of axillary and pubic hair. The Wassermann reaction was negative, and there was no history of syphilis or any other infection to which the bradycardia could be attributed.

Peri-orbital Nocturnal Neuralgia cured by Peptone.—Mme. Nageotte-Wilbouchewitch showed a girl, aged 8 years, who had been subject to severe attacks of periorbital neuralgia, which woke her up regularly at 4 a.m. The condition, which had followed an attack of influenzal broncho-pneumonia, was cured by the administration of peptone for two months in doses of 0·50 grm. twice daily before meals.

Painful Polymorphous Dermatitis with Predominance of Bullæ.—MM. G. L. Hallez and Zuber showed a female infant, aged 5 months, in whom the polymorphous character of the lesions, the eosinophilia, absence of fever, intense pruritus and pain, and the subacute, not to say chronic, course suggested the diagnosis of Brocq's painful polymorphous dermatitis. The speakers stated that this form of dermatitis, which was less uncommon in childhood than was formerly supposed, might in exceptional instances develop shortly after birth, as in cases reported by Bowen, J. Hallé and Brocq. These authors stated that in half the cases under fifteen years of age pruritus was absent.

Loss of Water by the Lung in the Healthy Infant and in the Infant suffering from Cholera infantilis and Ordinary Diarrhœa.—MM. A. B. Marfan and H. Dorlencourt, as the result of their investigations, found that in normal children the average loss of water by the lung in ten minutes was 0·0318 grm. per kilo of body-weight, while in cholera infantilis the loss of water was increased by more than a third, and in ordinary diarrhœa, without any toxic or infective symptoms, the loss diminished by more than a half.

Insufficiency of the Soft Palate accompanied by several other Malformations.—MM. Apert and Bigot showed a boy, aged 8½ years, whose pronunciation suggested that he had a perforation of the palate. On examination, however, no such lesion was found, but the palate was very short and the uvula very small. On the other hand, the distance between the posterior border of the soft palate and the posterior wall of the pharynx was much increased, suggesting that the soft palate was too small to carry out its function of obturator to the pharynx. Deglutition took place normally at present, but up to the age of three years regurgitation through the nose was frequent.

This form of malformation of the soft palate was described in 1892 by Lermoyez, who collected twelve cases, including one of his own. Since then Castex and Egger in France had published three cases, and Gutzmann in Germany twenty cases. In all these cases the palatal condition was usually the only malformation present. Gutzmann, however, quoted a case of Gluck's, in which there was a flattening of the side of the head and thorax with a congenital cicatricial depression at the right buccal commissure. In the present case the palatal insufficiency was associated with a number of other malformations, namely, extreme brachycephaly, camptodactyly of the left ring finger and the second toe on both sides, absence of the xiphoid cartilage and an undescended left testis. The intelligence was normal. The child had been born at full term, and delivery had been normal. The parents were elderly, the father being fifty-three and the mother forty-two when the child was born. The Wassermann reaction was negative. There

was no similar case in the family as in some of the recorded cases. Some improvement was effected by making the child blow out candles, blow soap-bubbles, and training him to speak correctly.

Intercostal Zoster following Tuberculous Pleurisy with Effusion.—M. G. Schreiber reported two cases in children—a girl, aged 14 years, and a boy, aged 15 years—who developed intercostal zoster in convalescence from tuberculous pleurisy with effusion, but the zoster was on the same side as the effusion in one case and on the opposite side in the other. This seemed to indicate that if the tubercle bacillus was responsible for the occurrence of the eruption, as seemed probable, zoster was not a local complication, but a manifestation of a generalised tuberculous infection. Zoster might sometimes be the first sign of tuberculous infection, and therefore appear before the pleurisy.

Abstracts from Current Literature.

Acute Infectious Diseases.

Diphtheria in the newborn (*'Bull. Acad. de Méd.,'* 1921, lxxxvi, *p.* 191).—**S. Brindeau** reports an epidemic of nine cases of diphtheria among newborn children in a lying-in hospital. All the infants were healthy and born at full term. All presented the same clinical symptom—coryza. All but one died in a few days in spite of diphtheria antitoxin, which was used in five cases. Preventive injections of antitoxin were given, no case of serum disease developed, and the outbreak, in which the newborn were exclusively attacked, was brought to an end without it being necessary to close the service. J. D. Rolleston.

Faucial diphtheria in the first months of life (*'Le Nourrisson,'* 1922, x, *p.* 44).—**G. Blechmann** and **M. Chevalley,** after quoting Rolleston's statistics (*vide* British Journal of Children's Diseases, 1917, xiv, *p.* 64), report a fatal case of faucial and nasal diphtheria in a female infant, aged 1 month, who ten days before the onset of diphtheria had shown a negative Schick reaction. They conclude that in the infant too much confidence should not be placed in passive immunity of maternal origin, and that the ordinary methods of prophylaxis should be rigorously carried out in crèches and nurseries, whether the Schick reaction be performed or not. J. D. Rolleston.

Diphtheria of the glans penis (*'Arch. f. Kinderheilk.,'* 1921, lxx, *p.* 112).—**P. Bode** reports a case of diphtheria of the glans penis in a boy, aged 6 years, associated with faucial diphtheria. The disease ran a favourable course without any complications. There were two possible modes of infection of the penis in this case, viz. either by direct transfer of diphtheria bacilli by the hands to the penis, or by the urine. Conradi and Bierast found diphtheria bacilli in the urine in 54 out of 155 cases of diphtheria (*vide* British Journal of Children's Diseases, 1912, ix, p. 508), and Beyer obtained further results in all the 19 cases of diphtheria which he examined. J. D. Rolleston.

Diphtheria with bilateral orchitis during the serum manifestations (*'Arch. de méd. des enf.,'* 1921, XXIV, *p.* 744).—**G. Blechmann** and **J. Stiassnie** report the case of a boy, aged 6½ years, with a severe attack of diphtheria treated by large doses of antitoxin, who developed an extremely painful bilateral orchi-epididymitis at the time of the serum manifestations. With the fall of the temperature and subsidence of the other symptoms of the serum disease the scrotum diminished in size and the pain subsided. The writers regard the orchi-epididymitis as a hitherto undescribed localisation of the symptoms caused by serum. J. D. ROLLESTON.

Case of primary anal diphtheria (*'La Pediatria,'* 1921, XXIX, *p.* 318).—**M. Mallardi** reports the case of a boy, aged 22 months, suffering from pain and irritation in the genitals and perinæum. The anal orifice was red and swollen. On separating the folds there were two small fissures with grey exudation at the bottom. There was fever, constitutional disturbance, and glandular enlargement in the peri-anal region. Microscopical examination showed numerous streptococci and bacteria having the morphological characteristics of diphtheria bacilli. Recovery ensued on treatment with antidiphtheritic serum and dressings of ichthyol and hydrogen peroxide.
VINCENT DICKINSON.

Wound-diphtheria responsible for a diphtheria outbreak (*'Arch. of Ped.,'* 1921, XXXVIII, *p.* 732).—**A. D. Kaiser.**—The patient was a girl, aged 16 years, admitted to a general hospital with the diagnosis of empyema. There was little relief from the operation. Repeated small hæmorrhages took place through the incision and the patient presented the picture of toxic absorption. When the dressings were changed one day a dirty grey exudate from which diphtheria bacilli were cultivated was found about the tubes. 18,000 units were injected intravenously, but death took place from cardiac failure a few days later. The necropsy revealed membranous pleurisy and pericarditis, and smears and cultures from the pleural and pericardial membranes and pericardial fluid yielded typical diphtheria bacilli. The case was responsible for three cases of diphtheria among the nursing staff, one of which was laryngeal, three in the adult ward and two in children, while cultures of the throats of thirty children who were negative on admission were positive in eight cases. J. D. ROLLESTON.

The heart in diphtheria (*'Journ. Amer. Med. Assoc.,'* 1921, LXXVII, *p.* 765).—**C. H. Smith** examined 242 cases of diphtheria, and found that 72 per cent. had no evidence of cardiac disturbance apart from an initial tachycardia of 135 to 172. The remaining 28 per cent. after an interval of several days showed certain vagaries of the pulse, viz. sinus arrhythmia and sino-auricular block in 65 per cent., premature contractions in 20 per cent., none of these three phenomena being accompanied by symptoms or signs of circulatory embarrassment. The remaining 15 per cent. consisted of high grade heart-block, which was very sudden in onset, and was associated with very urgent cardio-circulatory symptoms with death within 36 hours. Auricular fibrillation was not observed. J. D. ROLLESTON.

Diphtheria bacillus carriers; results of re-examination of apparently negative cultures (*'Johns Hopkins Hosp. Bull.,'* 1922, XXXIII, *p.* 110).—**B. C. Marshall** and **C. G. Guthrie** show that when dealing with throat cultures from diphtheria carriers the customary practice of making

single or repeated examinations within the first twenty-four hours may not yield all the information available and may lead to erroneous conclusions. Of 549 cultures which were negative on the first examination, 52, or 9·47 per cent., were positive on examination after an additional incubation of twenty-four hours. Similar results were obtained in 1912 by Knebel, who found that among 576 negative cultures from diphtheria convalescents, 55, or 9·54 per cent., were positive when examined after twenty-four hours. Thus out of a total of 1125 cases in which the cultures were negative after examination within twenty-four hours, 107, or 9·51 per cent., were positive on re-examination. J. D. Rolleston.

The application of bacteriological studies to the prevention of diphtheria (*'Public Health,'* 1922, xxxv, *p.* 203).—**F. W. Andrewes** states that different strains of the bacillus vary in toxicity, one strain killing a guinea-pig in twenty-four hours, and another in four days. A freshly-isolated strain is either markedly toxic or wholly non-toxic. Strains isolated from carriers are fully toxic or wholly non-toxic. Intermediate properties are rarely met. There is a lack of evidence that non-toxic strains ever become toxic again. There is little relationship between toxicity and the severity of the attack. Intense toxicity may be found in bacilli recovered from mild cases of diphtheria. The susceptibility of the individual determines the onset and severity of the attack. If one-fiftieth of a unit of antitoxin exist in a c.c. of blood the subject is immune to the disease. 80 per cent. of infants are passively immune at birth, but this gradually disappears till at three years of age only 28 per cent. react negatively to Schick's test. After this immunity again rises till at eighteen years of age 80 per cent. are actively immune, perhaps due to sub-infection during childhood, not sufficiently severe to set up recognisable diphtheria. The percentage of immunity is far higher among the lower classes. There is no certain means of ridding the carrier of his bacilli, but disappearance often occurs after removal of enlarged tonsils and adenoids. Virulent carriers should be isolated in the country. Re-infection of one case by the other would be avoided by open-air treatment, and removal of those whose swabs had become negative to another part of the building. The wide application of the Schick test and immunisation is strongly advised, but resort to legal compulsion is not recommended. Christopher Rolleston.

The Schick test (*'Lancet,'* 1922, i, *p.* 312).—**T. E. Dickinson** used in 1200 cases Burroughs and Wellcome diluted toxin and as a control the toxin heated to 75° C. Injection was made into the forearm and was followed immediately by a raised white urticarial wheal. Inspection of the forearm was made 24 hours, 72 hours and 10 days after injection. The positive reaction is characterised by a slightly raised and infiltrated area 10–30 mm. in diameter, varying in colour from a deep pink to a dark dusty crimson. The reaction is at its height 48 to 72 hours after injection. At the end of the first week there is brown pigmentation and scaling. The latter may vary from fine desquamation to massive exfoliation. The reaction was delayed in 154, or 14·1 per cent.; 147 were delayed one day, 6 four days, and 1 five days. In 10, or 0·9 per cent., abnormal pigmentation occurred after the tenth day. Massive scaling occurred in 2 and vesiculation in 3. The largest number of positive reactions occur in the fourth year of life, which agrees with the age-incidence of these diseases and the mortality figures. The pseudo-reaction is an anaphylactic phenomenon which reaches its

maximum in 20 to 30 hours. It is of more vivid red, begins to fade before 48 hours, is more infiltrated, and only occasionally leaves a faint pigmentation. There is often a well-marked pink zone round it. It is larger than the positive reaction. The reaction due to the control is of this nature. A reaction in both arms means a pseudo-reaction, which fades equally in both arms. In a combined reaction the control arm clears up quickly while the other goes through the various stages of the positive reaction. Pseudo-reactions in 10 per cent. of the cases persist for more than a week. They are more than twice as common in those over than in those under 15 years of age, and among normal persons than those suffering from scarlet, enteric and puerperal fevers and erysipelas. The administration of anti-streptococcic serum probably inhibits the development of a positive Schick reaction. Scarlet fever predisposes slightly to diphtheria. Toxic cases gave a very low percentage of positive reactions—19 as compared with 47·38 of all scarlet fever cases. Vaccination increases by five times the frequency of pseudo-reactions. There is a high pseudo-reaction rate among enteric and erysipelas cases and a low one in puerperal fever. Infancy diminishes both the positive and pseudo-reactions. Cases treated with anti-diphtheritic serum gave a very high percentage of pseudo-reactions. Members of the same family tend to give the same positive or negative reactions, but do not agree in their pseudo-reactions. CHRISTOPHER ROLLESTON.

Schick's reaction in infants (*'Riv. di Clin. Ped.,'* 1921, XIX, p. 621).—**M. Flamini** investigated the Schick reaction in a foundling hospital in 145 children at the breast, 125 weaned infants aged from 1 to 3 years, and 190 nursing women. The percentage of positive reactions was 17 in infants aged 3 to 4 months, 29 in those aged 4 to 8 months, 36 in those aged 8 to 12 months, 46 from 1 to 2, and 50 from 2 to 3. The ages of the nursing women ranged from 17 to 30, most being about 20. Positive results were obtained in 60, or 32 per cent. There was no relation between the mother and child or between the nurse and the suckling as regards the presence or absence of diphtheria antitoxin, but if the suckling had antitoxin in its blood it was not inherited from the mother nor acquired from the nurse through the milk. J. D. ROLLESTON.

The management of a diphtheria outbreak in a private school (*'Journ. Amer. Med. Assoc.,'* 1921, LXXVII, *p.* 1714).—**E. C. Fleischner** and **E. B. Shaw**, who describe the management of a diphtheria outbreak in a private school of 150 patients, draw attention to the high percentage (65 per cent.) of positive Schick reactions in boys from 8 to 18 years of age. In view of the fact that these boys came from a stratum of society in which diphtheria was not prevalent, this suggests the possibility that repeated exposures to the disease played a *rôle* in the development of natural immunity. J. D. ROLLESTON.

The carrier of diphtheria bacilli from the fever hospital standpoint (*'Med. Officer,'* 1922, I, *p.* 91).—**H. R. Harries.**—Distinction has to be made between clinical diphtheria, some other pathological condition, plus saprophytic diphtheria bacilli, and the healthy "carrier." This point can now be eliminated by the application of the Schick test. A positive Schick reaction means that the patient is susceptible to the toxin of diphtheria, a negative, that he has sufficient antitoxin in his tissues to neutralise any toxin produced by diphtheria bacilli growing on his tonsils or elsewhere.

The toxin used must be active and recently diluted, a control must be used, the needle must be fine and sharp, and the test must be intradermal. No anti-diphtheritic serum must be given for at least six weeks before the administration of the test. Cases not presenting clinical signs of the disease are divided into the two following groups: Group 1—positive swab, (*a*) Schick positive, (*b*) Schick negative; Group 2—negative swab, (*a*) Schick positive, (*b*) Schick negative. Group 1 (*a*) is treated with antitoxin; Group 1 (*b*) is not. Group 2 (*a*) is also treated by antitoxin, but in Group 2 (*b*) antitoxin is not required. Three hundred and twenty cases of scarlet fever were swabbed and tested by the Schick test, with the following result: Negative swab and negative Schick 65·7 per cent.; positive swab and negative Schick 9·6 per cent.; negative swab and positive Schick 19·9 per cent.; positive swab and positive Schick 5·4 per cent. Twenty-five per cent. of these cases were therefore susceptible to diphtheria as compared with 33 per cent. in Park's series. Nurses giving a positive Schick should not nurse cases of diphtheria. Carrier cases have to be dealt with whatever the result of the Schick reaction. Virulence tests must be applied. In 161 convalescent and carrier cases 79 were virulent, 62 were avirulent, and 20 were of the Hoffmann variety and non-virulent. Patients with avirulent bacilli and giving a negative Schick reaction can be discharged at once from fever hospitals. Cases can be divided into those with (*a*) healthy, (*b*) unhealthy throat. Those in class (*b*) often re-infect saprophytically those in class (*a*). Harries discharged those in class (*a*) without swabbing and no "return" cases occurred. The only "return" cases occurred from class (*b*), which had been sedulously swabbed. No methods have yet been found which will free "carriers" from the bacilli.

CHRISTOPHER ROLLESTON.

Diphtheria control (*'Med. Officer,'* 1921, II, *p.* 275).—**B. Carey.**—In Massachusetts the death-rate from diphtheria has fallen from 43·3 per 100,000 to 15·4 during the last 20 years, but there has been very little improvement during the last 10 years of this period. The morbidity rate is still very high, varying from 283·4 per 100,000 as a maximum to 154·8 as a minimum. This failure is ascribed to (1) incomplete application of methods of proved worth, such as the Schick test and immunisation; (2) late diagnosis and late antitoxin treatment; (3) delay in receiving medical attention. Twenty-five per cent. of the undue incidence is due to recurrences in the same communities, and infection may be carried more readily by children of the pre-school age period. Several of the State institutions have been completely Schick tested followed by active immunisation. Two cities now maintain Schick clinics. Five thousand Schick tests have now been administered in Massachusetts, and only one susceptible person has developed diphtheria after a full course of toxin-antitoxin treatment. In one institution for boys in which outbreaks were frequent all the scholars were Schick tested and immunised, with the result that no further outbreaks have occurred. During an epidemic all contacts are treated by antitoxin, which produces passive immunity for 3 to 5 weeks. Later the contacts are Schick tested and the susceptibles immunised. Swabs are examined at once, and in 60 per cent. of the cases a positive diagnosis can be made. At 3- and 8-hour intervals cultures are examined and 90 per cent. can be reported. A final examination is made after 24 hours, confirming all previous diagnoses.

CHRISTOPHER ROLLESTON.

Schick test and diphtheria toxin-antitoxin immunisation (*'Public Health,'* 1922, xxxv, *p.* 219).—**T. E. Dickinson** states that at the Monsall Fever Hospital 12 Schick reactors among the nursing staff were treated. Three injections were given at weekly intervals. The mixture consisted of toxin just under-neutralised by antitoxin. Ten, or 83 per cent., of the nurses gave a negative Schick reaction one and a half to three months after injection. Two reacted violently, prostration, and fever to 103° F., being recorded. They had previously shown pseudo-reactions, and in such cases the dosage is reduced to 0·1 c.c., 0·2 c.c. and 0·5 c.c. for the first, second and third inoculations. The work could be carried out through the infant welfare and school medical services. One thousand Schick and control tests can be made at a cost of £20. The immunisation of 1000 persons entails an outlay of £300. CHRISTOPHER ROLLESTON.

Schick's reaction (*'Arch. de méd. des enf.,'* 1921, xxiv, *p.* 624).—**J. Comby**, in a review of the literature, acknowledges the value of the recent work on this subject but points out that Schick testing and active immunisation should not be the work of the general practitioner, requiring as they do bacteriological knowledge, constant recourse to the laboratory, delicate though simple instruments, and a minute technique. Systematic employment of the reaction is easy in a hospital where experts are available. Outside a hospital the investigation of carriers, Schick-testing, etc., should be undertaken by a municipal department of hygiene.

J. D. ROLLESTON.

Scarlet fever following thumb-sucking (*'Münch. med. Woch.,'* 1921, lxviii, *p.* 1679).—**R. Bloch** reports a case of surgical scarlet fever in an infant aged 18 months due to sucking its thumb, as the result of which a gaping wound had developed. The case fulfilled the conditions laid down by Hoffa in 1887, viz. development of the eruption at the site of the wound after the appearance of lymphangitis, swelling of the tonsils and submaxillary glands, and typical desquamation. Parents should be warned of the danger of infection in the case of every wound—even a trivial breach of surface caused by sucking—as scarlet fever is as likely to arise from this source as from drinking unboiled milk. J. D. ROLLESTON.

Prolonged infection in scarlet fever (*'Public Health,'* 1922, xxxv, *p.* 151).—**W. B. Young.**—A boy, B. N—, aged 7 years, developed a scarlet fever rash on June 20, 1921. His brother, D. N—, had septic scarlet fever on March 21, was discharged from hospital on June 3, 1921, and a day or two later developed severe tonsillitis. On June 10 his tonsils were removed. He came in contact with his brother on June 10. On July 28 both brothers went to the seaside and were joined by their brother, O. N—, who travelled down with the F— family, consisting of a lady and two boys, on July 29. On August 9, eleven days after their contact with B. N— and D. N—, I. Q— and O. N— developed mild scarlet fever. Another of the F— family, N. F—, and D. N's— mother developed scarlet fever. On August 27 Mr. N— returned home and was joined soon after by D. N—, who on September 17 journeyed with him to see friends. Mr. N— returned on September 19 feeling seedy, with a sore throat. No rash was detected. On September 20 D— was sent home, as cases of scarlet fever had occurred in the relations' home. D— was sent to a fever hospital and was found to have a thin mucoid nasal discharge. A Mrs. M—, living in another quarter of the town, developed scarlet fever. She had been lodging with a Mrs. A—,

who had been a servant in the N—s' house and who had been visited by D. N— on September 10. Two or three days later Mrs. A—s' child developed what was termed "German measles" and was nursed by Mrs. M—. The boy D. N— infected nine persons and his infectivity lasted over 26 weeks.

CHRISTOPHER ROLLESTON.

The tongue in scarlet fever ('*Journ. de Méd. de Paris*,' 1921, XL, *p.* 294).—**N. Cojan.**—Four distinct stages may be seen, and their recognition is of great value when the eruption is scanty or lacking. (1) The tongue is elongated, bright red at the point and edges, while the dorsum is covered with thick, white fur. This lasts two to four days. (2) The fur lessens from before back in the form of a **V**. (3) Towards the seventh or eighth day the whole tongue is raw, red or scarlet—the raspberry tongue. The papillæ show prominently. (4) The tongue ceases to be pointed, loses the scarlet colour and has a varnished appearance, becoming normal about the fifteenth day.

J. PORTER PARKINSON.

Miliaria in scarlet fever ('*Thèses de Paris*,' 1920–1921, *No.* 167).—**R. Barthe** states that miliaria in scarlet fever is rare below the age of three years, and is infrequent in the adult except in the puerperal woman. Its presence at the onset of scarlet fever may be regarded as an indication of an intense rash which will take a long time to disappear. Owing to its frequency at the onset of scarlet fever in children it possesses some diagnostic value, and in the presence of a doubtful eruption should be regarded as in favour of scarlet fever.

J. D. ROLLESTON.

Orchitis in scarlet fever ('*Il Policlinico*,' 1922, *Sez. Prat.*, XXIX, *p.* 155).—**Medi**, who reports a case of suppurative orchitis in a child aged $2\frac{1}{2}$ years, states that only three previous cases of genital complications in scarlet fever have been recorded, viz. one by Henoch in which inflammation of the epididymis and tunica vaginalis occurred on the nineteenth day of disease, a similar case described by Horteloup, and a third case described by Aucuña of a boy, aged 9 years, who developed slight bilateral orchitis with inflammation of the tunica vaginalis on the eighth day of disease. Unlike Medi's cases none of these ended in suppuration.

J. D. ROLLESTON.

Peritonitis as a complication of scarlet fever ('*Amer. Journ. Dis. Child.*,' 1921, XXII, *p.* 307).—**E. C. Dunham** has collected ten cases of peritonitis complicating scarlet fever from the literature of 1900–1919, including a personal case in a girl, aged 5 years, cured by operation. The diagnosis in four cases was confirmed by operation or by the necropsy. A streptococcus was grown from the pus in two cases. Blood cultures were not reported in any of the cases. The peritoneum is probably infected by the blood-stream in these cases.

J. D. ROLLESTON.

Purpura fulminans during convalescence from scarlet fever ('*Journ. Amer. Med. Assoc.*,' 1922, LXXVIII, *p.* 165).—**G. McConnell** and **H. L. Weaver** state that purpura fulminans is a rare condition, but that a relatively large number have followed scarlet fever, as is shown by the fact that J. D. Rolleston and T. McCririck in 1910 collected 16 instances of the kind (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1910, vii, p. 58).

Since then McConnell and Weaver have collected 7 more cases, including one of their own in a girl, aged 6 years, whose history was as follows: On the fifteenth day after the onset of scarlet fever, and on the seventh or eighth day of normal temperature, the temperature rose to 104°F. and the left foot became discoloured. Ecchymoses appeared on the body in the course of 48 hours, the urine and stools became bloody, and on the fourth day after the appearance of the first ecchymosis death took place. In addition to the skin ecchymoses the autopsy revealed hæmorrhages in the bladder, intestine and brain. Microscopical examination showed that the discoloration was not due to gangrene, but to intestinal hæmorrhage following infective thrombosis.

J. D. Rolleston.

Relapses in scarlet fever (*'Arch. de méd. des enf.,'* 1921, xxiv, *p.* 471). —**V. Hutinel** and **M. L. Nadal** attach much importance in the causation of relapses in scarlet fever to the co-existence in the early stage of the disease of some other more or less severe infection, the influence of this association being to modify the process of immunisation. They record 10 cases of relapse in cases of scarlet fever associated with some other infection, viz. lobar pneumonia in 3 cases, acute articular rheumatism in 1 and a surgical infection in 6.

J. D. Rolleston.

Diagnosis of scarlet fever (*'Deutsche med. Woch.,'* 1921, xlvii, *p.* 1428). —**U. Friedemann** attaches considerable diagnostic importance to Widal's hæmoclasic crisis, which he has hitherto found present in all cases of scarlet fever, sometimes from the first day of the disease and lasting well into convalescence. Hitherto with few exceptions it has proved negative in all other infectious diseases. The test is carried out as follows: The patient takes his last meal on the night before at 9 p.m. and at 8 a.m. in the morning drinks 200 c.c. of milk. The leucocytes are counted before and after at intervals of 20 minutes. A diminution of 2000 is regarded as a positive reaction, but it is usually much greater in scarlet fever.

J. D. Rolleston.

Prophylactic immunisation against smallpox (*'La Pediatria,'* 1921, xxix, *p.* 638).—**V. Tripputi**, from his observations during an epidemic in Palermo, 1920–21, found that Jennerian immunisation was a sure prevention from smallpox infection. Variolisation by injecting the contents of a variolous vesicle not infected with secondary germs gave rise to a mild reaction, which was efficacious in preventing infection, and was useful in this respect for a community in which a case of smallpox accidentally occurred. During an epidemic all newly born infants should be vaccinated. Vaccination practised in families in which a case of smallpox occurred did not unfailingly preserve all the members from variolous infection but only the greater part of them. If the disease breaks out it is not influenced by the development of the vaccinal pustule, which follows its own course independently of the action of the variolous infection. The smallpox is not influenced by the appearance of the vaccinal reaction. The interpretation of this fact is obvious, since the brief period between the inoculation and the outset of the variola is insufficient for the production of immunity.

Vincent Dickinson.

Torticollis from typhoid myositis (*'Arch. Lat. Amer. de Pediatr.,'* 1921, xv, *p.* 28).—**F. Bazán** records a case of a mild attack of typhoid fever in a girl, aged 5 years, complicated by torticollis due to myositis of the trapezius which ended in sclerosis. The child was predisposed to the complication by the fact that a year previously she had had an attack of torticollis of eight days' duration, and that she was also the subject of inherited syphilis, as shown by a positive Wassermann reaction.

J. D. Rolleston.

Restriction of influenza epidemics in schools (*'Brit. Med. Journ.,'* 1922, i, *p.* 433).—**A. I. Simey** and **J. Eyre** relate that severe epidemics of influenza affected Rugby School, 260 out of nearly 600 boys being affected, with a loss of 5000 school days. Inoculation was advised, but only 123 out of 570 were treated in the holidays. The following vaccine was administered, the number after each variety indicating millions. Pneumococcus (50), *Micrococcus catarrhalis* (25), streptococcus (10), *B. pneumoniæ* (50), *B. influenzæ* (10), *Staphylococcus aureus* (200). Fourteen days afterwards the following vaccine was given: Pneumococcus (100), *Micrococcus catarrhalis* (75), streptococcus (50), pneumobacillus (100), *Bacillus septus* (100), *B. influenzæ* (30). The doses of the various components are small when compared with those of the Ministry of Health. 233 boys received both vaccines and only 7·3 per cent. were attacked by the disease; 123 boys received one, two or three doses at home and 41 per cent. were infected; 160 were not inoculated at all, and 130 of these, or 81 per cent., developed the disease; 50 received the initial vaccine, and 43, or 84 per cent., contracted the disease, illustrating the fact that the first dose renders the individual more susceptible. Altogether 405 received one or more doses of vaccine, of whom 113, or 27·9 per cent., were subsequently attacked, compared with 81 per cent. among the uninoculated. Christopher Rolleston.

Sequels of influenza (*'Glasg. Med. Journ.,'* 1921, i, *p.* 267).—**M. F. G. Main** thinks that influenza is a potent factor in initiating many illnesses or in aggravating disabilities already present. A boy, aged 10 years, on recovering from influenza began to complain of thirst and polyuria, and sugar appeared in his urine. There was no history of diabetes in the family. Under treatment his condition greatly improved, and he was discharged free from glycosuria and able to take a fairly generous diet. Another boy, aged 11 years, when suffering from a mild attack of chorea contracted influenza. The chorea became much worse. J. Allan.

Intra-dermo reaction in the early diagnosis of pertussis (*'La Pediatria,'* 1921, xxix, *p.* 337).—**E. Modigliani** used the following preparation: A loop of pure culture of the Bordet-Gengou bacillus is suspended in 1 c.c. distilled water with the addition of a few drops of a 3 per cent. solution of toluol. This is placed in an incubator for twenty-four hours at 37° C., or for greater security forty-eight hours; the organisms will then be dead and their bodies broken up. Two drops of this solution are injected in the same way that tuberculin is used. A similar preparation of *B. coli* was used as a control. The test was made on 50 children suffering from various diseases, but free from pertussis; on 38 children from 5 months to 6 years of age, with characteristic signs of whooping-cough; 10 children recovered from whooping-cough; 3 children living with other infected children. In the first series there were no positive reactions; there were never any reactions with the

B. coli preparation. The children with pertussis all gave a positive reaction, which was most marked from seven to fifteen days from the beginning of the illness. In children recovered from the disease, or when it had lasted over two months, the reaction was negative or very feeble. The most remarkable fact was that there was a positive reaction in the three children living with other infected children, and who had not yet had any characteristic symptoms. After a few days the symptoms of pertussis made their appearance.

VINCENT DICKINSON.

Infantile amœbic dysentery (*'La Pediatria,'* 1922, xxx, *p.* 1).—**L. Spolverini** describes 5 cases occurring in children from $2\frac{1}{2}$–12 years of age. They were mild and almost chronic. The diagnosis was problematical until the stools were examined and the organism found, especially as two of the cases occurred in Rome, where dysentery is not common. The stools were often formed, abundant, composed of well-digested material, and from time to time contained more or less large lumps of mucus and sometimes bright blood. The author draws attention to the strict necessity of sterilising the child's clothes and bed-linen from a prophylactic point of view.

VINCENT DICKINSON.

Reviews.

CONFÉRENCES PRATIQUES SUR L'ALIMENTATION DES NOURRISSONS. By P. NOBÉCOURT. Third edition. Paris: Masson et Cie, 1922. Pp. 316. Price 18 fr. net.

IN the seven years that have elapsed since the publication of the last edition of this book (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1914, xi, p. 332.—ED.), fewer advances have probably been made in the subject of infant feeding than in any other branch of medicine of equal importance, owing, doubtless, to the war, which absorbed the attention of the doctors in the belligerent countries. Consequently comparatively few changes and additions are to be found in this new edition, especially as Prof. Nobécourt's aim is not so much a complete exposition of all modern teaching on the subject, as a lucid presentation of the essential principles involved in a correct dietary. In the first thirteen chapters of the book the various problems connected with infant feeding, both natural and artificial, and of weaning are dealt with exhaustively, almost exclusively from a practical point of view, the next two being devoted to a discussion of various laws and regulations affecting the welfare of nursing mothers, wet-nurses, and suckling infants, as well as a clear *résumé* of the position as regards syphilis and the infant. The last five chapters deal with the feeding of infants in various pathological conditions, this section being substantially the same as in the previous edition of the book, in which they were first incorporated. The volume contains forty-five illustrations and is furnished with an adequate index, a further excellent feature being the short summary at the end of each chapter.

The whole book is exceptionally well balanced and clearly written and is well worthy of the author's international reputation. E. M.

HAUTKRANKHEITEN UND SYPHILIS IM SÄUGLINGS- UND KINDESALTER. Ein Atlas herausgegeben von Prof. Dr. H. FINKELSTEIN, Berlin, Prof. Dr. E. GALEWSKY, Dresden, Dr. L. HALBERSTAEDTER, Berlin. Berlin: Julius Springer, 1922. Price in Germany, 260 marks; in England, 1040 marks.

THE writers remark in their preface to this "Atlas of Skin Diseases and Syphilis in Infancy and Childhood" that the appearances presented by skin diseases and syphilis in children differ in many respects from those usually seen in the adult. As these peculiarities have not hitherto received sufficient attention in dermatological text-books and atlases, the present volume, which is the joint work of a pædiatrist and dermatologists, has been undertaken with the object of supplying this want. The work consists of 77 pages of text containing a brief account of the skin diseases met with in childhood, and 123 coloured plates, which, with the exception of two provided by Neisser from the Breslau Skin Clinic, are from the collections of Profs. Finkelstein and Galewsky. The plates include illustrations of the acute exanthemata, diphtheria of the skin, serum eruptions, impetigo, scurvy, and the various forms of eczema, tuberculosis of the skin and ringworm, as well as rarer conditions, such as epidermolysis bullosa hereditaria, erythrodermia desquamativa and xeroderma pigmentosum. The different varieties of vaccinal eruptions are portrayed, together with the deplorable effects of intentional or accidental vaccinia in children suffering from eczema. Twenty plates are devoted to syphilis. The writers remark that acquired syphilis in children, which was previously a comparatively rare occurrence, has lately become more frequent.

The clear and concise descriptions in the text and the excellence of the plates should recommend this atlas to every pædiatrist. J. D. R.

ÜBER KLINIK UND EPIDEMIOLOGIE DER PERTUSSIS. By Dr. D. POSPISCHILL. 180 pages. Berlin: S. Karger. Price 35 marks.

THE author of this volume is, in respect of clinical experience, well qualified to write on pertussis. He is physician-in-chief to a large hospital for infectious disease in Vienna, and in the course of his nineteen years' office in that institution Dr. Pospischill has had under his personal observation 25,000 cases of whooping-cough, and carried out some thousands of post-mortem examinations in children dying from that disease.

This monograph, therefore, which deals with the clinical aspects and the post-mortem appearances of the disease, should not only abound in valuable observations, but should also grip the attention of the reader. We regret to say, however, that the work before us has not realised our expectations. The author devotes some 160 pages to the clinical and morbid anatomical features of the disease, in which he lays particular stress upon the alleged lasting effects of pertussis upon the subsequent health of the child. He believes that the results of infection with the disease make themselves felt after many years of apparent health. He finds that nearly all children with a positive Pirquet test have had pertussis. Dr. Pospischill also claims to be able to diagnose present or past pertussis from the presence of a zone of peculiar *râles* at the base of the lungs. In the remaining nineteen pages the author deals with treatment, in which he expresses great faith in the good effects of open air upon the progress of the disease.

We find no mention of the various blood changes produced by the disease, nor of the value of vaccine therapy from the point of view of prophylaxis and treatment. This omission is greatly to be deplored; with such

an abundance of material at his disposal, one ought to be able to come to some definite conclusions regarding the value or otherwise of the newer methods of treatment. Nor can we congratulate the author upon the manner in which he conveys his information. Indeed, we have no hesitation in saying that this work is a model of the manner in which a book should not be written. The style is heavy and uninteresting, and there are no headings to relieve the monotony of reading pages after pages of descriptive matter clothed in an abundance of padding. The work, it is true, contains a number of observations (some of which are of undoubted and others of doubtful value), but these are so submerged in an ocean of verbosity that only patient and skilful diving can succeed in bringing them to the surface. The absence of an index renders it useless as a reference book and the price is prohibitive. Goethe once remarked that the Germans have the special ability "to make knowledge inaccessible." We think that Dr. Pospischill can, in spite of his nationality, lay very good claim to this distinction.

W. M. F.

Technique Chirurgicale Oto-Rhino-Laryngologique. Par E. J. Moure, G. Liébault, et G. Canuyt. *Prem. fasc.* L'Oreille et ses Annexes. Paris: Librairie Octave Doin, 1922. Pp. xii + 362. 228 figures in the text and one coloured plate.

This new addition to an already large literature of the ear, nose and throat is a noteworthy one. It demonstrates well the two antithetical features of French medical and surgical publications—most abominable binding in the flimsiest of paper covers, with splendid detail in the contents, a contrast which might be imitated with advantage in Great Britain. French text-books which are intended for practical use by students and practitioners never permit possible misapprehension of directions as to technique in examination or operative procedure. Anything in which the reader might be misled in studying the text is always clearly illustrated by photograph, drawing or diagram executed with such clearness as to be of real assistance. This is eminently the case in the work under review, and although there are at least twenty out of the 228 figures which might have been discarded as needless, this redundancy is on the right side. The directions contained in the first chapter on the examination and therapeutics of the ear are very complete and profusely illustrated. They are followed by a chapter dealing with the surgery of the auricle. This should be of interest to the pædiatric otologist. Whilst the authors deprecate too much zeal in the endeavour to rectify deformities of congenital origin, the best methods are well described and figured.

The section upon the surgery of the mastoid is excellent, both as regards the acute and the chronic conditions. It contains one paragraph upon mastoiditis in the child and the infant. The authors make the statement that the diagnosis is not generally established until peri-mastoid swelling has made its appearance. We cannot wholly subscribe to this opinion, although in some cases it may be correct. In operating upon infants the reader is warned as to the high situation of the antrum, and admonished as to the necessity of great care and gentleness. The advice is excellent, but might have been rendered even more valuable had mention been made of injuring the facial nerve by a too careless incision. Nor is there any warning given of the necessity for conserving function whenever possible.

The present volume is, of course, merely the first of several. If, however, succeeding numbers are up to the same standard, the complete work will be one of high excellence. M. Y.

Les Néoplasmes. Revue exclusivement consacrée à la littérature, à l'étude et à la thérapeutique des tumeurs et des cancers paraissant tous les deux mois. 1922. Janvier, tome i, pp. 1–32. Paris: Vigot frères. Abonnements: France et Colonies 15 fr.; Étranger 18 fr.; Le numéro 3 fr.

In 'Les Néoplasmes' we welcome a new contemporary devoted entirely to the literature, study and treatment of new growths, which is under the direction of Joseph Thomas for medicine, Paul Delbet for surgery, and Charles Schmidt for radium and X rays, and will appear every two months. This first instalment contains a review of the red blood-corpuscles in cases of malignant disease by Prof. Albert Robin, and from the large number of references contained in this carefully classified account provides a valuable summary of our present knowledge. In the other original article, on the relative value of X-rays, radium, and surgical excision in carcinoma of the tongue, Paul Delbet draws on his own experience, and divides the cases into those of the base of the tongue, in which the operative risk is so high that he no longer employs it, and carcinoma of the body and tip of the tongue, in which he prefers excision followed by the intensive application of X rays to the alternative of the insertion of radium tubes into the growth. This number also contains 34 abstracts arranged alphabetically under the names of the authors. H. R.

The Journal of Metabolic Research. Edited by Frederick M. Allen. Published monthly by The Physiatric Institute, Morristown, New Jersey, U.S.A. 4°. January, 1922, vol. i, No. 1. Pp. 163, with many illustrations and diagrams. Price $10 per year.

The first number of this new journal contains six papers from the Hospital of the Rockefeller Institute for Medical Research (New York), and one from the Physiatric Institute (Morristown, New Jersey). Five of the articles are concerned with the pathology of diabetes, and two with experiments on carbohydrate metabolism and diabetes. Of considerable general medical interest from the ordinary clinical point of view are two articles: (1) "Nervous Influences in the Etiology of Experimental Diabetes," by Dr. F. M. Allen; and (2) "The Influence of Glucose Ingestion on Diuresis and Blood Composition in Non-Diabetic and Diabetic Persons," by J. W. Sherrill and H. J. John. From his experiments in regard to nervous influences, Allen concludes that no influence of emotion upon the production of diabetes could be demonstrated. In one instance a violent general traumatism seemed to activate a patent diabetes, but the effect was transitory. The Claude Bernard piqûre seemed to be a genuine factor in producing diabetes in one predisposed dog. Complete separation of a pancreas remnant from its original nerve supply failed to give rise to diabetes or any demonstrable lowering of assimilation. It also failed to affect either the occurrence or the rate of hydropic degeneration in the islands of Langerhans; this degeneration ran parallel with the course of the diabetes the same as when the nerve supply was left undisturbed.

The papers should each one of them be read in the original by those interested in the experimental study of diabetes mellitus and metabolism. The printing of the text and the quality of the plates are excellent, and the

journal will doubtless meet with a good reception. The editor, in his introductory remarks, writes: "The journal is intended to serve for publication of the results of original research. As it occupies a border zone between the laboratory and the clinic it should be of interest to laboratory and clinical investigators, and to such medical practitioners as wish to keep in touch with fundamental scientific progress in metabolism." F. P. W.

A Comparative Study of the Health of Missionary Families in Japan and China. By William G. Lennox, M.D., Department of Economics, University of Denver, U.S.A. Pp. 44.

More than a year ago the author published a paper on the health of missionary families in China, the present pamphlet being a supplementary study of a similar investigation among missionaries in Japan, including a comparison of these results with the health of a similarly constituted group of persons in the United States.

The information, which was collected by means of a *questionnaire* sent to the 375 missionary families in Japan, of whom one half replied, is of wider interest than might at first be supposed, and of such a varied character as to preclude even the briefest *résumé* in the space at our disposal. Among the principal facts elicited are the evidence that children in Japan show a death-rate far below that of children in China, and below even the selected group in America, the figures for the various groups being—for missionaries in China, 60; for the selected group in America, 48; and for missionaries in Japan, 43. The remarkably low death-rate of infants and children in Japan, comparing well with the figures of 42 for professional classes in England, is, as Dr. Lennox points out, offset by the excessively high fœtal death-rate, 134 of each 1000 pregnancies among missionaries resulting in miscarriages or stillbirths, as against 117 in the American group. Two-thirds of these miscarriages have been ascribed by the wives to general conditions of over-work and under-health, Dr. Lennox emphasising the evident need for better professional care of the mothers during pregnancy and confinement, as also the fact that, in view of the large percentage of deaths of children from difficult feeding and malnutrition, the problem of a proper diet urgently presents itself for solution. Diphtheria is nearly three times more prevalent in Japan than in China or America, the author suggesting that the children, especially those for whom diphtheria antitoxin is not immediately available, should be given the Schick test, and if found not to be immune should be made immune by toxin-antitoxin injections.

Many other points of interest are dealt with, Dr. Lennox's work undoubtedly forming a useful contribution to the subject of pædiatrics, not only affecting the health of missionary workers in Japan, but also conducing towards a better understanding of the broader aspects of the problems involved. E. M.

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